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PRACTICE OF MEDICINE

VOLUME VII

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EDITED BY

FREDERICK TICE, M.D.

PROFESSOR OF MEDICINE AND CLINICAL MEDICINE, AND
HEAD OF THE DEPARTMENT OF MEDICINE,
UNIVERSITY OF ILLINOIS, COLLEGE
OF MEDICINE

FOREWORD BY

M. W. IRELAND, M.D.

SURGEON-GENERAL, U. S. ARMY

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SECTION X: DIGESTIVE SYSTEM DISEASES OF THE DIGESTIVE SYSTEM

INTRODUCTION

Great interest has always been manifested in the study of diseases of the digestive tract as an important and integral part of clinical medicine. It has, however, been in recent years only that an intensive study has been directed to these affections, and that the specialty now known as gastro-enterology has been recognized as an important subdivision of clinical medicine.

This specialty really dates its origin as far back as the beginning of the nineteenth century when the use of the stomach-tube was first recorded. There has been much discussion as to the priority of the discovery of the stomach-tube, and the view has been generally accepted that this instrument is of English origin, and that the two English surgeons, Jukes and Bush, are its inventors. This claim is based on an article published by Jukes in the London *Medical Repository* of 1822 under the title, "Description of an Apparatus for Removing Poison from the Stomach Invented by Mr. Jukes, Surgeon"; however, it is but a simple matter to demonstrate that the credit of this discovery is due to Dr. Physick, who published his original paper October, 1812, in *The Eclectic Repository*, iii, 3, under the title of "Account of a New Mode of Extracting Poisonous Substances from the Stomach, by Philip S. Physick, M.D., Professor of Surgery in the University of Pennsylvania"; this is ten years prior to the appearance of Jukes' article. He declares that he employed the tube in washing the stomachs of twins three months old who had accidently been given an overdose of laudanum by their mother. He utilized a large catheter for this purpose. One of the children was saved; the other died. Physick states that the idea of washing out the stomach with a syringe and tube in cases in which quantities of laudanum or other poisons had been swallowed occurred to him at least twelve years before, and he had constantly for many years recommended it in his lectures. In the year of 1809 his nephew, Dr. Dorsey, practiced lavage in a case, but the patient had swallowed the poison twelve hours before he was called, so that he did not succeed in saving life. But the tube was soon forgotten, and was not generally employed until after 1869, when Kussmaul suggested its use in the treatment of gastric dilatation.

In an extremely valuable paper, "Über die Behandlung der Magenerweiterung durch eine neue Methode mittlest die Magenpumpe," Kussmaul presented a most original method of treatment which laid the foundation for an exact mode of diagnosis in gastric affections. He tells of the difficulties besetting the treatment of advanced forms of dilatation of the stomach due to narrowing and closure of the pylorus and how by means of the stomach-pump, it is possible to empty the stomach completely and, if its elastic and contractile powers have not been entirely lost, perhaps even give it the tone to contract to a certain degree. The stomach-pump would not only make it possible completely to evacuate these acrid acid masses but would permit the washing and cleansing of the diseased mucous membrane.

Although most enthusiastic over this new method of treatment Kussmaul realized its limitations. He recognized that only where slight constriction of the pylorus existed was he able to cure cases of dilatation of the stomach by lavage. In cases of malignant stenosis of the pylorus and marked cicatricial contraction relief only could be afforded, and not cure. Thus by this epoch-making work of the great master Kussmaul, not only was a new method of treatment given us, but it also furnished the impetus for the study of gastric diseases according to modern scientific methods and led to the stomach tube, not only as a new method of treatment but also of diagnosis.

A year later (1870) Jurgensen pointed out the disadvantage of the stomach-pump, as recommended by Kussmaul. The pump is difficult to cleanse and in fact its use is superfluous. Jurgensen here introduces the principle of siphonage into the practice of gastric lavage, which entirely eliminates the use of the pump in this mode of treatment. The soft-rubber tube is also here recommended, and Jurgensen fully explains the method of introduction of the stomach-tube, the position to be assumed by the patient, as well as the practicing of siphonage in freeing the stomach of its contents. This contribution must be classed as a distinct advance in the therapeutics of gastric diseases. Jurgensen utilized the rubber tube terminating in a perforated ivory bulb. The bulb was guided by means of a wire stylet. Four years later (1875) Ewald called attention to the fact that any rubber tubing having sufficient resistance could be introduced into the stomach without the use of a guide.

While Kussmaul drew attention to the therapeutic value of the stomach-tube, it remained for Leube at the Rostock Naturforscher Versammlung in 1871 to point to its value as a diagnostic measure.

Twelve years later (1883) Leube established his test-meal as a means of determining the fact that a normal stomach should be empty seven hours after the ingestion of a meal of water, soup, steak, and bread. It was not long after (1883) that Leube developed his well-known scale of the digestibility of various foods, which he earnestly advocated as a means of diet in the treatment of ulcer of the stomach. This work is most valuable, pointing as it does to the importance of diet in gastric diseases, and marked a distinct advance in the therapeutics of diseases of the stomach.

It was Riegel (1881) who demonstrated the difficulty encountered in obtaining gastric juice by stimulating the gastric secretion by means of ice water, according to the method of Leube; he therefore advised the withdrawal of the gastric contents after his well-known test-meal of soup, beefsteak, bread and water.

Not many years after (1885-1886) Ewald and Boas recorded their investigations on the appearance of free hydrochloric acid and lactic acid in the stomach contents, and also drew attention to the other important factors associated with the chemistry of gastric digestion. They accepted the height of digestion as the period best adapted for withdrawal of the gastric secretion for examination. Ewald fully described his test breakfast, and presented evidences of its advantage as well as its importance in the diagnosis of gastric disease.

A further advance in the diagnosis of gastric affections was made by Guenzburg's (1887) announcement of his test for the detection of free hydrochloric acid and by Uffelmann (1880) for lactic acid.

Following the classical observations of Beaumont and similar experiments upon individuals having gastric fistulæ made by Richet and by Uffelmann, Penzoldt's work on the study of the digestion of food in the stomach published in 1893 is extremely valuable. In these remarkable investigations Penzoldt describes the work undertaken by his students upon themselves. Each individual ingested certain quantities of food and removed the same at varying intervals with the tube (fractional withdrawal) in order to determine the length of time each foodstuff remained in the stomach, assuming that the digestibility of food depends largely upon the time required for the stomach to empty itself. These experiments form a valuable and exhaustive investigation upon the digestibility of the various foods, and a most important addition to the study of modern dietetics.

The greatest credit is due to Pawlow (1902) and his pupils for the many new facts discovered relating to the physiology of gastric digestion. By means of a blind pouch from a portion of the stomach separated from the stomach itself by a septum of mucous membrane and communicating with the outside world by a fistula, he was enabled to study carefully the secretion of gastric juice.

Coincidently with this study of the gastro-intestinal tract abroad, numerous clinical observations were published in this country relating to this subject. Preceding the "Pioneer American Physiologist," Beaumont, whose remarkable experiments upon Alexis St. Martin, published in 1823, are epoch-making, it is interesting to note that another American investigated the physiology of digestion thirty years before the publication of Beaumont's work. This fact was brought to light by Prof. L. B. Mendel, who refers to a thesis entitled "An Experimental Inquiry into the Principles of Nutrition and the Digestive Process," submitted to the faculty of the University of Pennsylvania in 1803 by Young, a Marylander.

Among the interesting clinical observations published in the early part of the nineteenth century is the publication of Jameson in 1825, who reported a case of stricture of the esophagus illustrating the employment of a very ingenious set of instruments for the dilatation and cure of this affection.

Diaphragmatic hernia which has elicited so much interest in recent years was well described by Edward Cornelt in 1825 in a case report of a child ten months old.

We find an anticipation of later discoveries upon the relationship of bacteria and parasites and their toxins to disease in an article by Charles Frick in 1851 on "The Pathology of Epidemic Dysentery." The writer suggestively remarked: "If we consider attentively the collection of symptoms, and note the great mortality attendant upon them, even in the first few days, we cannot but be struck with the fact that the cause of death is not sufficiently evident; that the severity of the local disturbance is inadequate to produce a fatal result, and the idea constantly obtrudes itself, that some morbid poison in addition to the local disease has an agency in producing the extreme prostration and subsequent fatal termination."

It was about the middle of the nineteenth century that a new era dawned in American medicine, when experimental investigation began to forge into prominence. Up to this time the work in gastro-enterology had been largely of a clinical character, but the work of Beaumont on the physiology of digestion, which had been heralded abroad, had sown the seed of enthusiasm and zeal for research on this side. Jackson in 1854 confirmed Bernard's observations concerning the digestion of fats, which had been disputed by Bidder and Schmidt, and Lehman and Frerichs.

John Dalton about this time described fully the nature and functions of the gastric juice based upon a series of experimental studies; and in 1857 the same author contributed a most admirable and instructive article on "The Constitution and Physiology of the Bile." He presents in detail his researches upon the comparative study of bile in different animals with particular attention to its chemistry. He demonstrated that bile passes into the intestines of carnivorous animals for at least twelve hours after meals, and that it is discharged most abundantly immediately after feeding.

In 1860 Flint called attention to the relationship between anemia and atrophy to the gastric tubules, and expressed the belief that some cases of obscure and profound anemia are dependent upon degeneration and atrophy of the glands of the stomach. It was twenty-six years later that the well-known paper of Henry and Osler on "Atrophy of the Stomach with the Clinical Feature of Progressive Pernicious Anemia" was published in which it was again pointed out that a primary atrophy of the gastric mucous membrane might occur, and the theory advanced that certain cases of progressive pernicious anemia depended upon profound alterations in the gastric tubules.

Of great importance, too, is the classic work of J. J. Woodward on "Diarrhea and Dysentery," published in 1879 in "The Medical and Surgical History of the War of the Rebellion." In this comprehensive volume of 869 pages Woodward gives a complete statistical review of the cases of diarrhea and dysentery occurring during the war, descriptions of the fatal cases, an account of the pathological changes observed, and a discussion of the treatment of these affections. There can be but little question that this extraordinary volume may be classed as one of the greatest works published in the field of gastro-enterology in America.

To Meltzer is due the credit of first having investigated the peristaltic movements of the esophagus. Associated with Kronecker in 1883, he demonstrated the incorrectness of the view, previously maintained, that the food is forced into the esophagus by the contraction of the pharyngeal muscles and showed that the contraction of the mylohyoid muscle shoots the bolus through the entire esophagus in the case of liquid or very soft food. When the food is semisolid or solid, however, Cannon and Moser demonstrated by means of the x-ray that the passage through the esophagus is accompanied by peristaltic contraction of its muscles. It was Meltzer, too, who in 1883 first noted the importance of the sounds of deglutition.

A work which did much to develop the entire field of gastro-enterology in this country is the excellent and comprehensive production of Welch in Pepper's "System of Medicine," 1885, comprising contributions on ulcer and cancer of the stomach and dilatation. This work is in many respects the most exhaustive ever published on these subjects. The entire literature is reviewed and new statistics prepared from a large series of cases, the whole forming a complete monograph, which has helped to form the foundation for the study of these conditions according to modern methods.

Great credit is due Reginald Fitz for his masterly and epoch-making work on acute pancreatitis, published in 1889, in which he treats of the etiology and symptomatology of this affection and its relationship to other diseases. He discusses the significance of pancreatic hemorrhage, the cause of sudden death in acute pancreatitis, and the relationship of acute pancreatitis to fat necrosis.

It was but little more than a decade later that Opie pointed out the frequent association of cholelithiasis and disease of the pancreas and also demonstrated the lesions in the islands of Langerhans in diabetes.

W. J. Mayo in 1887 was the first to direct attention to the affection, "Acquired Diverticulitis of the Large Intestine," while our knowledge of appendicitis is almost entirely due to the contribution of American observers and investigators.

The first case reported in this country was by Walcott Richards of Cincinnati in 1837. Dr. Edward Hollowell of Philadelphia reported a similar case in 1838. These are 2 out of a total of 4 cases reported in the United States for a period of thirty years. During the early part of the fifth decade the vermiform appendix became a subject of frequent discussion. George Lewis of New York in the late fifties published a classical paper which was the first systematic contribution to the literature of the appendix in this country. Forty-seven cases are collected and tabulated.

William Parker of New York reported 4 cases in 1857 and William T. Bull presented an analysis of 67 cases in 1873. It remained for Reginald Fitz in his scholarly paper published in 1886 to clear up many obscure questions concerning this subject, to establish the name appendicitis, and to bring about a correct understanding of the morbid conditions affecting this organ and emphasizing their importance.

Van de Warker of Syracuse was the first to call attention in this country to the operative treatment of tuberculous peritonitis. This was in 1887. Three years later Osler contributed a valuable monograph on this subject, in which he reported the result of histological investigation on the process of healing after simple abdominal incision. He concludes "that tuberculous peritonitis is often a latent affection, localized in the peritoneum, which may even run its course without special symptoms; that as in other local tuberculous processes, there is in this a natural tendency to healing which takes place more frequently than has hitherto been supposed; and that statistical evidence shows laparotomy to be in many cases a palliative and in some instances a curative measure."

Booker in 1890 was the first to contribute a careful bacteriological study of the summer diarrheas of infants. This suggests the mention of Herter's excellent work in 1907 on "Bacterial Infection of the Digestive Tract," which comprises a complete review of the subject together with the results of much original research.

The first case of amebic dysentery recorded in America was reported by Osler in 1890. This was soon followed by the report of cases by Musser, Stengel and Dock, and a most elaborate monograph upon this subject by Councilman and Lafleur in 1891. The latter publication is a most exhaustive treatise upon the characteristics of the amebæ and the pathology of the disease.

Much work upon the nature of bacillary dysentery and the character of the organism producing it has been accomplished by American investigators. Flexner in 1900 described the lesions associated with this disease, and the rôle played by the Shiga bacillus as the etiological factor. Duval and Bassett in 1902 first pointed out the association of the *Bacillus dysenteriæ* (*Shiga*) with the summer diarrheas of infants. Howland in 1904 gave an exhaustive description of the pathology of the Shiga bacillus infection. Flexner and Holt in 1904 reviewed this entire subject, presenting complete bacteriological and clinical studies.

Of greatest import is the work of Cannon on the physiology of digestion. This investigator has clearly pointed out the salient points regarding the movements of the gastro-intestinal canal. He called attention to the fact that the salivary digestion continues for a considerable period in the cardiac area and has shown, moreover, that when carbohydrates are ingested thoroughly, mingled with saliva, 80 per cent. more sugar is present at the close of an hour in this part of the stomach than in the pyloric portion, which establishes an important fact regarding salivary digestion. By means of the x-ray it was shown that the stomach is not emptied suddenly upon the completion of the gastric digestion, but gradually and intermittently, the presence of hydrochloric acid producing a relaxation of the pylorus.

Space does not permit a review of other advancements of gastroenterology in this country during recent years. One need only casually refer to a few achievements, chief of which follow: The development of roentgenology of the gastro-intestinal tract by Einhorn, Williams, Hulst, Cannon, Tousey, Pfahler, Pancoast, Baetjer, Cole, Carman, Mills and many others; the further development of esophagoscopy and gastroscopy by Chevalier Jackson; the introduction of the duodenal tube with its many possibilities in diagnosis and treatment by Einhorn; the disturbance of the law of contrary innervation as a pathogenic factor in diseases of the gall-bladder by Meltzer, which led to the introduction of the method of nonsurgical biliary drainage by Lyon; the application of fractional analyses of the gastric contents by Rehfuss and of the duodenal contents by Einhorn; the researches of Lockhardt, Phillips and Carlson pointing to the direct relation of certain motor activities of the stomach upon the relaxation of the pyloric sphincter, thus indicating the presence of a mechanical as well as chemical factor associated with this phenomenon; and the researches of Alvarez according to which the rhythmicity of the gastro-intestinal tract is graded downward from the mouth to the anus, that is the muscle segments nearest the cardia show the greatest tendency of rhythmic contractions, while those from the pyloric region are slower in starting, and the same rhythmicity is present as a gradient throughout the whole bowel progressively decreasing toward the lower bowel, and thus, according to Alvarez, the underlying gradient of tone is the essential factor in determining the direction of peristalsis.

With the increasing interest in gastro-enterology in this country it gradually became apparent that some organization was needed in which new problems might be discussed and interest fostered in the systematic investigation of the digestive processes.

On June 3, 1897, a small band of men met in Philadelphia and perfected plans for the organization of The American Gastro-enterological Association. This association has steadily grown and under its auspices much good work has already been accomplished. It has gathered together the physiologist, internist, and surgeon, all interested in this special branch of medicine, and its transactions represent in many respects the leading work accomplished in this specialty in this country during the past twenty-five years.

For the past few years, due to the great interest manifested by many clinicians in this special field of medicine, need was recognized for other associations, and on this account the Section of Gastro-enterology and Proctology of The American Medical Association and the Southern Gastro-enterological Association have been organized.

While the need for such associations must be recognized by all, it must constantly be borne in mind that many manifestations seemingly directly associated with the disturbances of the digestive tract, often protean as well as atypical in character, are due to diseases of organs distant from this tract, and in which the digestion is only secondarily involved, and that it therefore behooves us to constantly bear in mind that the digestive tract is but a portion of the entire body, and cannot be entirely separated from it, and consequently attention cannot be entirely limited to it alone. The gastro-enterologist must therefore necessarily be above all else thoroughly conversant with the various phases of internal medicine, and it is only in this way that the best results can be accomplished.

In conclusion, let the gastro-enterologist take to heart the words of the renowned author of the masterword in medicine: "You enter a noble heritage, made so by no effort of your own, but by the generations of men who have unselfishly sought to do the best they could for suffering mankind. Much has been done; much remains to do; a way has been opened, and to the possibilities in the scientific development of medicine there seems to be no limit."

Julius Friedenwald, A.M., M.D.,
Advisory Editor in Gastro-enterology.

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PRACTICE OF MEDICINE

VOLUME VII

SECTION X

DISEASES OF THE DIGESTIVE SYSTEM

CHAPTER I

DISEASES OF THE SALIVARY GLANDS

By Henry Joachim, M.D.

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GENERAL SYMPTOMATOLOGY

The salivary glands are six in number. Their secretion, mixed with that of the oral cavity, constitutes the saliva. The parotid, as well as the submaxillary gland, possesses one excretory duct, while the sublingual gland has several (Rivinus). Because of this adequate drainage, pathological conditions of the latter gland produce few symptoms. Disturbances of these glands usually manifest themselves by one or more of the following symptoms:

A. Excessive secretion, ptyalism, salivation, or sialorrhea.
B. Diminished or absent secretion, xerostoma or asilia.
C. Enlargement of the glands.
D. Pain.

Excessive Secretion or Ptyalism.—Etiology.—Inability to swallow the normal amount of salivary secretion results in an accumulation in the mouth, which is expectorated or drools down the side of the cheeks.

This condition is often confused with a genuine hypersecretion of the glands or ptyalism. Pseudoptyalism, as it has been called, occurs in coma (anesthesia), in facial or bulbar palsy, when the tongue and lips are involved, in tonsillar and peritonsillar swelling, retropharyngeal abscess, and in esophageal paralysis, diverticulum, and stenosis. It is a prominent symptom in dislocation of the jaw and cretinism. A true hypersecretion occurs as a psychical reflex in the following conditions: The sight or odor of food produces what is commonly known as watering of the mouth; joy, fright, worry, and anxiety are sometimes the cause; the condition has been known to occur during sexual excitement or during the orgasm; it is sometimes a prominent and early feature in the psychoses, as in general paralysis, melancholia and dementia precox. The sound of high, shrill, and disagreeable notes is sometimes a factor in the production of the condition. It is a common symptom in hysteria. It has been found in organic nerve disturbances, for instance, as the aura of an epileptic attack, in tabetic crises, in brain tumor, in hemiplegia, in trigeminal neuralgia (tic douloureux), encephalitis, chorea, migraine and paralysis agitans. Salivation from central causes is produced by stimulation of the nucleus salivatarius which is situated between the facial and motor fifth nuclei. The intermediary nerve of Wrisberg which later joins the facial and becomes the chorda tympani arises from this nucleus. Local mouth conditions, such as dentition, the manipulations of the dentist, various forms of stomatitis, alveolar abscess, etc., are common factors. Tobacco, spices, alcohol and bitter substances in general produce an increased flow. Of the general conditions in which this manifestation may be noted, the following may be mentioned: nausea, ovarian diseases, pregnancy, pancreatic disease, gastric hyperacidity (water brash), intestinal parasites, otitis media (chorda tympani stimulation), and hydrophobia. It is sometimes an indication of anaphylaxis. The reactions following the administration of mercury, pilocarpin or iodids are frequent examples. It may be a symptom in morphine addicts when the drug is withdrawn. The condition has been known to occur in pellagra. There are reports of cases of so-called salivary diabetes in which ptyalism with sugar in the saliva alternates with polyuria and glycosuria. It has been noted in uremia and as a reflex in angina pectoris.

SYMPTOMATOLOGY.—Because of the excessive secretion the patient is constantly obliged to swallow or drool. This interferes with speech. Sleep may be disturbed or interfered with if the patient sleeps with an open mouth and wets his pillow. If the mouth is closed and ptyalism exists, the saliva sometimes trickles into the larynx and produces a paroxysmal cough, which may be followed by an asthmatic attack in those subject to this disease. The skin about the mouth becomes red and irritated from the constant moisture. Sometimes a rhinorrhea coëxists or alternates with pytalism. The excessive secretion, if swallowed, produces gastric disturbances by diluting or neutralizing the gastric juice. If the salivation is extremely severe, it may be accompanied by constipation and oliguria. The glands sometimes become swollen and are the site of a sensation of heat. The sense of taste may be impaired.

TREATMENT.—Treatment depends upon the etiological factor. Symptomatically **atropin, opium** derivatives, and **bromids** may be useful. If the secretion of the mouth is excessively acid, **alkaline mouth washes** are indicated. As an astringent mouth wash dilute solutions of **alum in water** or **glycerin** have been recommended (10 grains [0.65 gram] to the ounce). Radiation of the parotid region may diminish the secretion.

Diminished Secretion (*xerostoma, asilia*).—Dry mouth is of frequent occurrence in febrile affections. It depends upon diminished salivary secretion due to lack of mastication and fluid intake, and to the loss of body fluids (cutaneous and pulmonary). Mouth breathing is an important factor. The administration of preparations of belladonna and atropin are a common cause of diminished salivary secretion. It occurs as a result of excessive drain of fluids, as in diabetes insipidus and mellitus, granular kidney, vomiting and diarrhea. It is a manifestation of senile atrophy of the salivary glands. The secretion is often psychically inhibited in anger and fear (dry throat). The rice ordeal as practiced in India for the detection of criminals depended upon the failure of the guilty individual to secrete saliva when given some rice to masticate. Dry mouth is a common complaint in hypertension and in the various neuroses. It is a frequent accompaniment in intestinal stasis, in the menopause, myxedema, in inflammations of the salivary glands, and in salivary calculus. It occurs after therapeutic radiation of the thyroid, tonsils, pharynx and salivary gland. It is also a symptom of paralysis of the chorda tympani nerve.

TREATMENT.—Symptomatically small doses of **pilocarpin, iodids, calomel**, etc., have been administered. **Galvanism** and the application of the violet ray over the glands has been of some benefit especially in cases of nerve origin. Frequent mastication is important in febrile stasis.

Enlargement of the Salivary Glands.—Swelling of the glands is due to:

(1) Physiological functions such as mastication, and the administration of pilocarpin. It occurs also as an anaphylactic manifestation (hyperemia). It is said that the parotid occasionally enlarges vicariously in amenorrhea.

(2) Inflammatory conditions (sialoadenitis), as in mumps, typhoid, malaria, sepsis, abscess, or syphilis, tuberculosis, actinomycosis, etc., and to mercurialization and plumbism.

(3) Retention of secretion due to obstruction of the salivary ducts by calculi, cicatrices, or inspissated secretion, or to compression of the ducts by tumors or exudates (tumor salivarium).

(4) Neoplastic conditions, i.e., the presence of chondromata, myxomata, sarcomata, etc. A leukemic infiltration may cause tumefaction. Echinococcus cysts may occur.

(5) Emphysema of the gland structure or surrounding tissues. This occurs in the case of glass blowers or players of wind instruments. It sometimes follows attempts at catheterization of the duct of Steno for the detection of calculi or after injury, either intra- or extra-oral. Gas bacillus infection of the parotid may produce parotid emphysema. This condition is known as a parotid pneumatocele. Mediastinal emphysema may manifest itself by a prominent crackling tumefaction in the region of the parotids.

Enclosed within the parotid gland are numerous lymphatic nodes which drain the eyelids, auricle, auditory meatus, etc. Focal enlarge-

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ment or intraparotid adenitis may be mistaken for parotitis or enlargement of the parotid.

Pain.—This is usually a sensation of tension more than of actual pain. Compression of the trifacial nerve may be the cause of severe pain, which is sometimes accompanied by facial flushings. Mastication may be painful or difficult, due to the involvement of muscles or to tumefaction of the glands.

SPECIAL DISEASES OF THE GLANDS

Inflammation of the Parotid Gland.—Etiology.—Infectious parotitis or mumps is discussed in another section. Inflammatory conditions of the parotid glands are most often produced by:

(1) Extension from the mouth—oral infection.

(2) Hematogenous or metastatic disturbances after abdominal operations, particularly after oöphorectomy. It may occur in any form of sepsis (septic endocarditis) or terminal infection.

(3) Contiguity with inflammatory conditions of neighboring structures (auditory canal).

Oral infection occurs most commonly in the course of the infectious diseases such as typhoid, scarlet fever, measles, and in conditions associated with coma or inability to take solid food by mouth. It is not uncommon during the prolonged rectal feeding of gastric or duodenal ulcer. Mercurialism most commonly produces an inflammation due to stomatitis. It may, however, irritate the gland directly, since mercury is excreted by the saliva. The same applies to lead, copper, and iodin. Obstruction of the salivary ducts may lead to stasis and infection. Parotitis occurs in the cachexia of carcinoma.

Symptomatology.—The symptoms produced by the implication of the parotid are pain and tension in the pre-auricular region. The ear stands out prominently. There is a feeling of warmth in the affected region. The movements of the head become restricted, and mastication and deglutition become painful, difficult, or impossible. There may be a distinct dryness of the mouth on the affected side. The condition is usually ushered in by chills and fever. There may be deafness and tinnitus. Delirium is sometimes a prominent symptom.

On examination one observes a prominence in the pre-auricular region and the overlying skin has a shiny and reddened appearance. There is a distinct filling out of the space that usually exists between the angle of the jaw and the mastoid process. On palpation the gland is found to be tender, brawny and indurated. Later, as suppuration ensues, fluctuation may be noted. There may be evidences of facial paralysis. On inspection of the mouth, an areola sometimes surrounds the opening of Steno's duct (Tresilian's sign). On pressure over the gland a drop of serous or purulent fluid can sometimes be expressed through the orifice of the duct. There is usually a high leukocyte count.

Differential Diagnosis.—Syphilis, tuberculosis and actinomycosis are difficult to differentiate. There may be involvement of both parotids giving the characteristic pear-faced appearance. Occasionally there is an enlargement of the buccinator or sucking pad of Bichat which is a fatty pad between the masseter and buccinator muscles. This may be mistaken for an enlargement of the parotid gland. One must exclude erysipelas, the edema of trichinosis, and the enlargement of the pre-auricular gland which occurs in Parinaud's conjunctivitis, leukemia, etc., and in connection with subcutaneous emphysema due to gas gangrene or mediastinal emphysema. Tumors and von Mikulicz's disease must be considered. The parotid inflammation may also be a secondary phenomenon in diseases of the auditory canal or ear.

Complications.—The condition may be complicated by:

(1) Suppuration.

(2) Rupture of a parotid abscess, producing an oral, auditory, or facial fistula.

(3) Extension into neighboring structures, the neck or mediastinum.

(4) Thrombophlebitis of the facial or posterior jugular veins.

(5) Meningitis.

(6) Gangrene from excessive tension between the layers of the fascia which enclose the parotid.

(7) Pancreatic involvement.

(8) Involvement of the ovaries, testicle, and breast.

(9) Facial palsy.

(10) Otitis media.

(11) Involvement of the temporo-maxillary joint.

(12) Necrosis of the jaw.

Treatment.—Prophylactically, treatment includes oral hygiene and the encouragement of frequent mastication during the course of any prolonged illness. When the disease has once made its appearance, the local application of an **icebag**, the administration of **salicylates** or **urotropin**, and the application of **iodine** preparations are indicated. The ingestion of large amounts of **fluids** must be encouraged. In the presence of suppuration it becomes necessary to resort to **incision** and **drainage**. Pilocarpine hydrochloride in doses of gr. 1/20 may be tried.

Course and Prognosis.—Secondary parotitis is always of bad prognostic significance, especially when it occurs in the course of typhoid, pneumonia, or post-operatively. It is usually an evidence of poor resisting powers. In favorable cases the mouth becomes moist.

Pathology.—The gland is hard and enlarged. The interalveolar fibrous tissue is infiltrated with the products of the inflammation. The epithelium of the ducts undergoes desquamation (stenosis). The process may advance to suppuration and gangrene.

Submaxillary and Sublingual Inflammations.—The etiology and pathology are the same as those of inflammation of the parotid.

Symptomatology.—In affections of these glands the pain is usually referred to the floor of the mouth or to the angle of the jaw. Movements of the tongue become painful and difficult. There is dysphagia. An enlargement is visible in the floor of the mouth or under the chin or rami of the jaws.

Complications.—The chief complication is the so-called Ludwig's angina, which is a purulent inflammation involving the floor of the mouth and secondary to sublingual or submaxillary suppuration. The jaw bone or the periosteum may become involved.

Salivary Calculi.—Etiology.—These calculi occur most often in the submaxillary gland. Predisposing causes are the *diathèse calculaire* of the French, or the arthritic diathesis of Wright. Mercurialization, foreign bodies (detached tartar from the teeth), salivary stasis, and the

FRIED FILLETS OF VEAL

INGREDIENTS :

thin slices of veal	2-3 ozs. lard
flour	salt, pepper

Method :

Beat the slices of veal till very thin, dip them in flour, seasoned with salt and pepper and fry them quickly in hot fat.

Time : 10 minutes.

PAPRIKA VEAL

INGREDIENTS :

thin slices of veal	top of 1 pint milk
1 onion	salt
2 ozs. margarine	pinch of paprika

Method :

Cut up onion into rings and fry in hot margarine till a golden brown. Sprinkle with paprika and salt ; add beaten slices of veal. Cook gently with the lid on, occasionally adding very little water, for about 1 hour. Add the top of the milk and let it simmer for another 10 minutes.

Time : 1 hour. If slices thin, 1 week's ration is sufficient for 4 persons.

FILLETS OF VEAL IN MILK

INGREDIENTS :

thin slices of veal	1 pint milk
2 ozs. margarine	salt, pepper

Method :

Fry the well-beaten slices of veal in hot margarine for 1 minute on both sides. Pour the milk over them, season with salt and pepper to taste and simmer gently until soft.

Time : ½ hour.

VEAL STEW

INGREDIENTS :

1 week's ration of veal	2 ozs. lard
1 onion	salt
1 tomato	paprika

Method :

Chop up the onion very finely and fry in hot fat till a golden brown. Add tomato and paprika to taste and meat cut into small cubes. Season with salt and cook it gently on a very low flame, with the lid on, only adding a very little water when necessary.

Time : 1 hour. Sufficient for 3-4 persons.

PORK

When the Butcher says : "There is nice pork this week."

Pork in itself is so tasty that you do not need to add much to it to produce a nice dinner. The only thing is, that being rather expensive, the same trouble as with veal, you have to eke it out very carefully to make the rations last a few days. As pork chops have to be cut rather thickly, they will not go a long way. If roasted whole you only get one really good hot meal out of it. My experience has been that the best value is the boneless pork you very often find at the butcher's these days. If you cut thin slices from this boneless meat you will find that 1 week's rations will last for 4 meals.

fingers or floor, decomposing and sticky foods. Acids and alkalis have the same effect. Pyorrhea and pus from an alveolar abscess may act in the same way. The abuse of tobacco and the use of strong condiments are not guiltless. Stomatitis with violent edema is reported by Dabney as due to eating Brazil nuts. Nursing mothers with stomatitis may infect their babies.

(*c*) *Thermal* irritants, such as very hot or cold foods or drinks, are familiar factors.

(2) *Condition of Tissues as Affected by the General State.*—General and wasting diseases may so lower vitality as to permit the action of the irritants mentioned, such as tuberculosis, syphilis, typhoid and the exanthemata. Chronic diarrhea, rheumatic fever, nephritis and gout are often complicated by stomatitis. Renshaw adds neuropathic (herpes) and metabolic influences.

(3) *Excretion of Saliva.*—The saliva may excrete irritants, such as mercury, lead, iodids, and phosphorus, but the process is then apt to be ulcerative.

SYMPTOMS.—Catarrhal stomatitis is most frequently seen in infants, whose first trouble is due to the pain that prevents taking nourishment. In such cases there is restlessness and more or less fever. In violent cases the mucous membrane is very red, swollen and hot. At first there is a simple, dry erythema, but a catarrhal process soon supervenes, with an abundant mucous discharge. In older children and adults there is no elevation of temperature, but after the teeth have appeared the gums may be very puffy and painful, bleeding easily at the slightest touch. The breath becomes unpleasant. In the more severe cases, the neighboring lymphatic glands are involved. The whole buccal lining may be affected without partiality. The true saliva is always diminished and is occasionally inhibited, but later on excessive secretion of mucus fills the mouth and drools from the lips of children and mentally deficient or careless adults.

DIAGNOSIS.—A mistaken diagnosis is impossible, though it is most important to determine whether the trouble is primary or secondary.

COMPLICATIONS.—The complications of primary cases are few and infrequent. Diarrhea is sometimes seen in infants and children. Vomiting may follow ineffectual efforts to eject the ropy mucus. Regional adenitis has been noted. Eczema of the chin may be due to the drooling excretion. Convulsions, also, have been reported. Superficial ulcers may arise from a violent process or too strenuous therapeutic applications. Catarrhal stomatitis is often associated with pellagra, sprue and acute infectious diseases—especially measles, scarlet fever and typhoid —and may depend upon gastro-intestinal derangements affecting the pharynx and tonsils, from which the inflammation may extend.

TREATMENT.—Its treatment is of the simplest character. The vast majority of cases get well if let severely alone. Harm is oftener done by too vigorous therapy. Any obvious cause should, of course, be removed. **A purge at the beginning** is rarely out of place. Until teeth appear, only the very gentlest applications should be made to the mouth.

Babies' mouths are oftener injured than helped by rubbing, and Bednar's aphthæ may be so produced. The pain is best relieved by the **local application of a 1-2 per cent. solution of cold soda bicarbonate or borate**, every half hour, while the infant is awake. This should be administered by means of a dropper or a swab of cotton. **Feeding at usual intervals** should be maintained with spoon or dropper, or even gavage, if sucking is too painful. If the trouble proves intractable after a few days, Dunn recommends that a **1 per cent. solution of silver nitrate** be employed with great gentleness once in 24 hours and the **mouth washed carefully with cold sterile water after each application. A pinch of borax** should then be introduced into the mouth. Older children and adults rapidly recover when the cause is removed. Goadby thinks mild acid lotion best followed by application of galvanic current, if obstinate. Osbourne recommends:

℞

Quininæ bisulphatis	.1	gram	(grain 1.543)
Acidi borici	5.	grams	(grain 77.16)
Syripi ipecacuanhi	25.	c.c.	(fʒ 6.76)
Glycerini	25.	c.c.	(fʒ 6.76)
Aquæ menthæ piperitæ	200.	c.c.	(fʒ 6.76)

M. Sig. Dilute one tablespoonful with an equal quantity of warm water and use on the tooth-brush or as a mouth wash.

This is one of the very best applications the author has ever used. Astringents (as witch-hazel) are necessary in chronic forms. Be sure to see that no nasal or pharyngeal obstruction exists.

PROGNOSIS.—The prognosis is only serious in marantic children whose difficult nourishment is thereby complicated. Repeated attacks interfere with dentition and promote decay of the teeth afterward. The condition also may become very trying in typhoid fever or other protracted diseases and interfere very much with the taking of food.

PATHOLOGY.—The pathology has been sufficiently indicated, though sometimes occluded mucous glands look like blackheads over the surface.

Aphthous Stomatitis

SYNONYMS.—Stomatitis herpetica, vesicular stomatitis, follicular stomatitis, maculofibrinous stomatitis.

The variety of synonyms indicates the numerous efforts to designate the characteristic features of this form, as well as the controversy that the interpretation of the lesions has elicited. Both Moss and Riesman consider herpes of the mouth a distinct condition. It is really a simple catarrhal stomatitis in which lesions, like subepithelial vesicles, surrounded by a red zone, have made their appearance.

ETIOLOGY.—No satisfactory etiology has ever been decided upon. Microörganisms of high and low degree have in turn been arrested on suspicion and discharged after a variable period of trial. It may be the result of nerve toxemia, which produces its effects in the mouth, on account of obscure local conditions there. It is popularly associated with

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"disordered stomach" and is often precipitated by dietetic imprudence in those predisposed. Gerstenberger considers it due to lack of water soluble vitamin B.

SYMPTOMS.—No age is exempt except, possibly, infancy. The disease begins with little reddish spots or aphthæ on the interior of the lips, cheeks, and tongue, though they may be found also on the gums and palate. These spots are extremely painful and interfere seriously with nourishment. The lesion soon becomes vesicular and is very rapidly converted into a yellowish, abraded spot, hardly deep enough to be called an ulcer, in uncomplicated cases. Sometimes it resembles a false membrane. Successive crops may appear, though the individual lesion does not last more than a few days and heals without scarring. The saliva is abundant and may flow from the mouth. Several aphthæ may coalesce, forming a large, irregular lesion. Constitutional disturbance is very slight or absent. The breath is not affected unless actual ulceration is added. It is a self-limited trouble and rarely exceeds a week in duration.

DIAGNOSIS.—The condition is readily recognized by inspection, though it has been confounded with foot and mouth disease, in which the vesicular lesions extend over the neighboring integument. In doubtful cases the previous history (such as ingestion of raw milk from diseased cows) must decide the case.

COMPLICATIONS.—Aphthous stomatitis may be complicated by ulceration and herpes of the lips and skin. It may be associated with neurasthenic states, pregnancy and lactation, and in the latter case it will probably prove obstinate. The first dentition is a specially favored period for this variety of stomatitis, which adds materially to the already sufficient difficulties of feeding at that time.

TREATMENT.—There is some diversity of opinion as to the proper therapy, but all agree that **nitrate of silver**—preferably, in the writer's experience, **the solid stick**—is most efficacious for the actual lesion. **Mouth washes and potassium chlorate** are considered unnecessary by Cabot, but advised by Dunn, who considers that the elimination of the potassium chlorate through the saliva is most salutary. Feeding should be carried out as in the simple form. Yeast, tomatoes and orange juice in the dietary will relieve according to Gerstenberger's theory.

PROGNOSIS.—The prognosis is good in the main, though the process may be most obstinate and recurrent in adults, who then often exhibit distressing complications, such as extensive herpes. Development may suffer, owing to the difficulty in feeding delicate children.

PATHOLOGY.—The pathology has not been agreed upon, though the presumption is in favor of a maculovesicular eruption, in which the lesions are modified by the location.

Thrush

SYNONYMS.—Mycotic stomatitis, soor, muguet, sprue, stomatitis hypomycetica.

ETIOLOGY.—This is a form of stomatitis due to a fungous infection, most frequently seen in children, but also in debilitated adults.

Oïdium albicans and *Saccharomyces albicans buccalis* divide the odium of causation. Castellani considers it due to many different fungi, chiefly of the Monilia Persom group. The biological position of the parasite is, as stated, still undetermined. It is a yeast or a mold, and may be either, depending upon the case, seizing on the delicate mucous membrane of the child's mouth, if the latter is at all injured. Transmission is afforded by infected fingers, nipples or utensils in practically all cases, and the disease may become epidemic in infant asylums. Debilitated adults are also likely to become victims.

Symptoms.—The first sign—though occasionally preceded by a simple stomatitis—is the appearance of small whitish spots on the tip and edges of the tongue and on the inner sides of the lips, where the mucous membrane is most liable to injury during the act of sucking. The spots grow in size, coalesce, and may line the entire inner surface of the mouth. The resemblance to flecks of curdled milk is striking, and the lesions are slightly raised above the level of the mucous membrane. The patches can be removed forcibly without always drawing blood, but the proceeding is very painful. Sucking, chewing and swallowing cause much suffering. The saliva is rather more scanty than in other inflammations of the mouth and is distinctly acid in reaction. This comparative dryness may be a predisposing factor to infection. The fungus may spread to the tonsils, pharynx and down the esophagus, and even invade the stomach. In one of the author's cases, where it complicated typhoid fever, the gullet was so obstructed as to necessitate gavage, which had to be repeated every six hours, so rapidly did the growth refill the channel. It was the cause of death.

Diagnosis.—The diagnosis is made by the microscopical examination of some one of the patches removed for the purpose. Specks of milk are thus readily distinguished. It is practically the only fungous form attacking the mouth in this way. Authorities are still divided as to the proper classification. For many years the mold, *Oïdium albicans*, bore the entire responsibility, but several authorities—notably Cabot and Dunn—doubt the evidence and insinuate that a yeast fungus may be actually to blame. Probably there is more than one parasite capable of producing the lesions. Complications occur chiefly by the extension to neighboring parts or, rarely, even by metastasis through the blood to the brain and kidney. Eczema of the nates too is not uncommon. It is more apt to be associated with exhausting diseases, such as diabetes, tuberculosis and typhoid, as stated above. Marasmic, neglected and dirty children are ready victims. It is rare in a healthy person of any age.

Treatment.—*Prophylactic.*—Cleanliness of the infant's mouth and of all feeding utensils is imperative. Rubber nipples and bottles should be boiled after each use. In the case of the breast-fed, the mother's nipples should always be washed with boric solution followed by boiled water, before each nursing. Patients suffering from the disease should be rigidly separated from the unaffected.

Curative.—As a curative measure, the best is the gentle persistent use of a **saturated solution of boric acid** after each feeding. In very obstinate cases, the areas may yield to the application of a **1 per cent. aqueous solution of formalin.** Should the esophagus become involved the tube used for gavage should be anointed with a saturated solution

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of boric acid. Minute doses of **calomel** have been recommended for this, but signally failed in the case referred to above.

Feeding with a spoon or dropper may become necessary, and is recommended by Dunn in every case, when the bottle is being introduced to infants.

PROGNOSIS.—The prognosis is good in ordinary cases, if treatment can be carried out thoroughly and general health maintained, but becomes very serious in the debilitated or when the fungus invades other parts, especially the esophagus.

PATHOLOGY.—The thrush patches grow on both squamous and cylindrical epithelium and are composed of mycelial threads and spores of the fungus, together with epithelial cells, bits of food, a few leukocytes and some fibrin. Food and medicine may discolor the patches. The threads grow between the epithelial cells and may even penetrate the squamous layer, rarely reaching and invading blood-vessels, through which metastasis, already mentioned, may take place. On cylindrical epithelium it cannot grow so readily, as there is only one layer of cells. It clings tenaciously and usually, though not necessarily, causes bleeding on efforts at removal. Some forms show only sporulation, like yeast, and others—the more common—have threads and spores.

Ulcerative Stomatitis

SYNONYMS.—Stomacace, phlegmonous stomatitis, putrid sore mouth, trench mouth, ulceromembranous stomatitis.

DEFINITION.—It is an inflammation of the mouth restricted to the possessors of teeth, and characterized primarily by ulcers at the margin of the gums and, later, extending to the other neighboring parts.

ETIOLOGY.—Predisposition is necessary and is found in dirty mouths with carious and neglected teeth. Undernourishment, debility and overexertion are influential. Mercury, lead and phosphorus are peculiarly prone to facilitate the trouble, for which, most probably, the fusiform and spirochete combination of Vincent is finally responsible, though absolute proof is lacking. The presumption is favored by the invariable presence of this combination, its contagiousness and its disappearance, when healing is complete. It is present in many normal mouths, which may be "carriers." Ancillary organisms with trauma may be necessary, also. The frequency among soldiers has been ascribed to the antiluetic treatment often administered to them, which makes the gums sore and therefore more vulnerable. Prolonged use of aspirin and antipyrin was incriminated by Bristow.

SYMPTOMS.—Stomatitis ulcerosa is attended by moderate fever, which, even in adults, may reach 102° F. (38.8° C.). There is also loss of appetite, restlessness and pain on efforts at eating. The gum swells at the tooth margin, turning a dark purple often, and protruding between the teeth, especially toward the front, in severe cases to the back also. The affected area is very tender and bleeds on the slightest provocation. The gum becomes softened and separates from the tooth, with a yellowish ulceration in the fold of tissue thus formed. These ulcers may spread irregularly on the gums to the lips, cheeks and edges of the tongue. The teeth may be loosened and even drop out. Salivation is profuse and the breath becomes horribly offensive, the odor clinging obstinately to the secretions of the mouth and being probably due to the necrotic tissue that is cast off under specific bacterial influences. The neighboring lymph-nodes are enlarged and apt to be tender. The process lasts about a week and then the ulcers begin to heal. Dunn does not consider simple cases contagious, though the disease may spread through asylums. There are several special varieties of this type which demand separate notice.

Clinical Varieties.—*Mercurial Stomatitis.*—Mercurial stomatitis was one of the first to be recognized, and is due to the overuse of the drug by susceptible subjects. Sometimes one dose is sufficient, and repeated small doses worse than a single large dose. It is known as *salivation* or *ptyalism,* from its most conspicuous feature. Pre-existing diseases, such as nephritis or cirrhosis of the liver, are thought to favor it, and pyorrhea certainly predisposes. It was held that the mercury injured the tissues in excretion and laid the gums liable to bacteria. It has no clinical peculiarities.

Ulceromembranous Form.—The ulceromembranous form—*trench mouth,* or *trench gums,* as it is commonly known—has come into great prominence, in consequence of its prevalence among the English soldiers in the great European War. According to Bowman, it is a spreading affection of the gums, especially around the last molar tooth, the tongue, mucous surface of the cheeks and tonsils. Wells contends that it begins in the pharynx or nose and spreads forward, but Barker and Miller consider that it always starts on the gum. Ulcers soon appear, which spread laterally everywhere except on the tonsils, where they go deeper. They are covered with a white friable membrane, which is much more easily removed than in diphtheria, though it then—as in that disease—leaves a raw bleeding surface. It strongly resembles scurvy, with easily bleeding gums, salivation and loosening, tender teeth. The last feature may become so marked that mastication is almost impossible and debility and emaciation may result. The breath is peculiarly fetid, and a corresponding horrible taste is complained of. The submaxillary and sublingual glands are almost always involved. The temperature may reach 105° F. (40.5° C.), though many cases do not exceed 102° F. (38.8° C.), and in most there is no fever. Severe mental depression is a conspicuous and distressing feature. Violent forms may eventuate in gangrene, and some so-called cases of noma may be of this nature. The hard palate has been perforated. It is clearly communicable and has been spread by careless dentists, though the more usual modes of conveyance are common eating and drinking utensils.

This form of stomatitis accompanies the sprue or psilosis of the tropics, which is characterized by a very obstinate and relapsing frothy diarrhea.

Recurrent Type.—Lach describes a recurrent type of ulceration of the mouth which is very obstinate. It is more common in women and follows acute infections—even chicken-pox in one case. Spots appear on the lips, cheeks, palate, gums and tongue, beginning as rounded ulcers with white sloughing bases and surrounded by an inflammatory area. They are very tender and slowly enlarge. On the lips the process resembles pemphigus vegetans. Tuberculin and Wassermann tests are negative. One case lasted ten years and finally yielded to nitrate of silver.

Gonorrheal stomatitis, though more common in the new-born, due to maternal infection, was, in a case reported by Mayhew, apparently contracted in a dental chair. There were hemorrhagic areas over the pillars and velum with a white, clinging exudate over the palate, cheeks, lower lip, dorsum of the tongue and oropharynx. There was an ulcer on the frenulum. The nose was not affected and there was no odor. No adenitis was appreciable. Relief was obtained in 48 hours by the use of a saturated solution of thymol and 2 per cent. nitrate of silver three times a day swabbed on. A raw bleeding surface was left which gradually healed.

DIAGNOSIS.—The diagnosis is easily made by inspection, but should be confirmed by cover-glass preparations from the exudate which, stained with carbol-fuchsin, readily reveal Vincent's organisms (the fusiform bacillus and spirochetes), in the trench form often associated with amebæ. Scurvy is to be distinguished by the absence of ulceration, the presence of petechial spots and much less offensive breath. Pneumococcic ulcerations do not give necrosis. The white necrotic gums, pain and smear preparations are conclusive, according to Goadby. Vincent's angina is a frequent and prominent consequence and the infection may even be found on the genitalia. Foot and Mouth Disease may show dusky ulcers on lips, gums and tongue after the vesicles burst.

COMPLICATIONS.—Of complications, noma, pyorrhœa alveolaris and adenitis deserve mention.

TREATMENT.—*Prophylactic.*—Prophylaxis consists in the proper care of the mouth and teeth. This is most effective. If the disease has developed, the removal of the cause, when possible, is the leading indication.

Curative.—Responsible drugs should be suspended and the mouth cleansed with antiseptic washes, such as **hydrogen peroxid or liquor antisepticus. Chlorate of potassium** is highly esteemed by many, both locally and by internal administration. If used in the latter way, a sharp lookout for its toxic effects, such as drowsiness, suppression of urine, heart weakness and even cyanosis, should be observed, especially in children.

The mercurial form is best met, after discontinuance of the drug, by **antiseptic washes** and by penciling the affected area with a **2 per cent. aqueous solution of chromic acid,** immediately neutralized with **soda bicarbonate in saturated solution.** If the pneumococcus is responsible, Flexner advises **optochin.**

The trench mouth requires isolation and yields readily to the following:

℞

Vini ipecacuanhæ	f℥ss (15 c.c.)
Glycerini	fʒi (3.75 c.c.)
Liquoris arsenicalis	q.s. ad f℥i (30 c.c.)

This must be used as a mouth wash and should also be gently but firmly swabbed on the ulcers twice a day. The teeth should be cleaned with a few drops on the brush, none of the mixture being swallowed. Bowman claims cures in 3 days by this means. Goadby recommends **chromic acid,** as in the mercurial form, but Ellis considers Bowman's method superior. **Arsphenamin applications, either in solution or suspension in glycerin** 3 or 4 times a day, are highly recommended, as is the Carrel-

Dakin solution also. Churchill's tincture of iodine followed by 20 per cent. silver nitrate or fresh sodium perborate, 1 dram to one-half glass of hot water every one-half hour, as a mouth wash is Stillman's reliance after touching the affected area with 10 per cent. neosalvarsan in glycerine.

The following spray is considered specific by Gunston:

℞

Vini ipecacuanhi	fʒ vi	(22.5 c.c.)
Glycerini		
Liquoris potassii arsenitis	fʒ v	(18.5 c.c.)
Hydrogenii peroxidii q.s. ad	f℥ xvi	(473.11 c.c.)

ṁ. Sig. Use as spray twice a day.

It is especially intractable if associated with anaërobes, when iodine and acid alcohol are the best application.

Driscoll treats all ravages of Vincent's combination with intravenous injection of 1 per cent. solution of antimony and potassium tartrate, freshly prepared and in freshly distilled sterile water. 5 c.c. is given slowly and in two or three days 10 c.c. may be similarly administered. He claims permanent cure after six doses at two or three day intervals. Be sure to get in the vein or results will be very painful and worthless. Mercuric cyanide 1 per cent. after cleansing with peroxide of hydrogen applied on cotton swab is recommended by Corby.

Prognosis.—Most simple cases get well in 10 days or 2 weeks, but the recurrent form is very obstinate and death is not unknown in the epidemics in the trenches. Ellis reports a fatal case in a servant maid 24 years old. In leukemia it is apt to be the final complication.

Pathology.—The lesion is very characteristic. It is a necrobiosis. Ulceration occurs after separation of the swollen border of the gum from the tooth, in which a purulent exudate is formed, and beneath which necrosis takes place.

Vincent's organisms are found in almost all cases, and generally combined with amebæ. When they are cultivated artificially with anaërobic precautions they emit the same foul odor that characterizes the breath of the victims. Ulcerative stomatitis resembles fetid bronchitis. The condition has not yet been communicated to experimental animals. The association of spirochetes and amebæ suggested Bowman's arsenic ipecac compound, given above, as a promising weapon of attack. They are obligate anaërobes, serophils and are Gram-negative. The bacillus resembles the *Bacillus diphtheriæ*, but is larger. It is fusiform and often has a transverse, colorless band around the middle. The spirillum is like that of relapsing fever and goes deeper into the tissues than the bacillus. It is slender, with pointed ends, and shows from five to eight wide irregular convolutions.

Stomatitis Gangrenosa

Synonyms.—Noma, cancrum oris.

Etiology.—The cause of this form of stomatitis has never been finally agreed upon. There may be several different microörganisms capable of the crime, under varying predisposing circumstances, most of which have been incidentally mentioned. Preponderance of testimony is in favor of Vincent's combination, which, with many contaminating associates, is the most frequent inhabitant of the affected area. The comparative absence of inflammatory reaction around the gangrene resembles the behavior of the tissues in Vincent's angina and is cited as corroborative of this view of the etiology. The observation—that ulcerative stomatitis, known to be due to this microörganism, precedes most cases—is a further point in its favor and has been produced in dogs by it. Cocci and bacteria of many other kinds throng the affected area, and peculiar filamentous forms have also been found.

All cases may not be alike and it may spread through hospitals like the old gangrene, to which they gave the name. It is an ancient affection, having been reported by Hollander Battus in the sixteenth century, when it must have been more frequent than now. The moist, foggy coasts of the northern part of Europe are more often visited by it than the southern and interior countries. It is rare in America.

SYMPTOMS.—This is a rare form of stomatitis and is characterized by a rapidly spreading destruction of the tissues. It involves the cheeks. gums, and alveolar process.

There is apt to be, first, a catarrhal or ulcerative stomatitis, which may be overlooked, but soon there is a severe swelling of the side of the face which is marked by "a fatty gloss and waxy pallor." Contrary to expectation, this is not very tender to the touch, but discloses a hard, diffuse infiltration. On examination of the mouth, a spreading ulcer will be found, with a greenish black center, emitting a foul odor which may have been the first thing to attract attention. The cheek is soon penetrated by a black fissured scablike mass and the destruction may extend to the eye or down on to the neck. The jaw may be destroyed and the teeth fall out. A zone of demarcation is not invariable before death. There is great variation in the suffering and constitutional disturbance. Some children continue playful, retain their appetites and have little fever; but more usually there is rapid toxemia, coma and death from exhaustion. The submaxillary lymph-glands are enlarged. Noma is almost always confined to one side of the face. It is occasionally found in the external ear and external female generative organs. Metastases in the stomach and colon have been described by McCorrick.

DIAGNOSIS.—The diagnosis is not difficult in a typical case. Rarely an ulcerated gum with abscess formation may simulate gangrene, but thorough evacuation and antiseptic treatment will promptly arrest the former.

SEQUELÆ.—Catarrhal pneumonia from inhalation of septic material, and nephritis, are serious sequelæ.

ASSOCIATION WITH OTHER DISEASES.—Stomatitis gangrenosa is itself a terrible complication of many infections.

Though sometimes a primary affection, it is more commonly associated with some infectious disease, especially measles, diphtheria, scarlatina, typhoid or dysentery, more rarely cerebrospinal fever, pneumonia and tuberculosis. Children between two and five years of age are the readiest victims—particularly girls.

TREATMENT.—*Prophylactic.*—Prophylaxis is limited to a careful hygiene of the mouth, especially in the infections already referred to as most likely to afford encouragement to the process. Ulcerative stomatitis may eventuate in this form and should accordingly be the more vigorously attacked.

Curative.—As soon as the disease is recognized, no time should be lost in its thorough extirpation by **surgery,** without awaiting a line of demarcation, which may never form. The **actual cautery** can follow the knife advantageously to insure completeness of removal. Afterward frequent inspection should be resorted to and any suspicious spots removed. It is apt to recur, so **plastic operations** should not be performed until such danger is surely over. **Intravenous and intramuscular injections of galyl** in 6 cgm. doses stopped the process rapidly, in one case reported by Peters, after surgery had failed.

Prognosis.—This condition is fatal in most cases; few patients recover (about 15 per cent.) and these become horribly disabled and disfigured. Promptness and thoroughness of treatment save some but, if pneumonia or nephritis set in, recovery is not to be expected.

Pathology.—The process is that of a spreading gangrene with slight or no inflammatory reaction in the surrounding tissues. There is no suppuration and little tendency to demarcation, although the latter may form and a slough be cast off.

Bednar's Aphthæ

Synonyms.—Ulcera pterygoidea, ulceration of the palatine angles.

Etiology.—It is a bilateral, symmetrical and superficial ulceration of the hard palate, far back. It is seen only in the newly born and in infants in the first weeks of life. Bouveret's ulcers in typhoid fever on either side of the fauces above the tonsils arise in much the same way.

Symptoms.—The ulcers are about the size of a pea, surrounded by a bright red zone and covered by a firmly adherent yellowish-white mass. They are very painful and interfere seriously with nursing. The lesions may not be absolutely the same size nor invariably bilateral; they may be joined by a bridge or may attack the raphé.

Diagnosis.—The diagnosis is easily established by inspection.

Treatment.—*Prophylactic.*—The prophylactic treatment consists in avoiding rough washing or wiping of the mouth—especially after thrush has occurred. A hard nipple or pacifier is an abomination.

Curative.—A 2 per cent. solution of **silver nitrate** daily, applied to the affected places, affords prompt relief.

Prognosis.—The prognosis is good.

Pathology.—Pathologically it is a pressure necrosis with secondary invasion by cocci, occurring in the parts of the mucous membrane which are most tightly stretched. The *cause* is a local injury from forcible and routine washing of the mouth or prolonged sucking on rubber nipples or pacifiers. It is certainly less frequent since these factors have been recognized than it formerly was.

Ulcer of the Frenum

Ulcer of the frenum is seen in subjects of whooping-cough. It is due to pressure of the tongue against the teeth during paroxysms.

Perlèche

Synonyms.—"Bad corners," foul angles of the mouth. Chelitis Migrans.

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DEFINITION.—This is an "impetigo" of the commissures of the lips and extends into the mouth. It is seen chiefly in children of school age, but adults are sometimes affected. It is very contagious and spreads through common drinking cups, towels and mouth kisses. According to Lane, it is much more frequent than is recognized, and has been epidemic in schools.

ETIOLOGY.—Lemaistre, who first—in 1886—described the disease, blames an anaërobic streptococcus for it, and Lane, in 1916, agrees with him, though he admits that several other kindred microörganisms may share the responsibility in individual cases. Small diplococci have been found but not proven causal. The germ disappears as healing progresses. The carelessness and untidiness of many children predispose them to the condition.

SYMPTOMS.—At first there is a mother-of-pearl color and a dryness in the corners of the mouth. The latter causes the patient to lick the affected part frequently. This disposition is always suspicious. The involved area then becomes macerated and a pellicle is discharged which leaves a reddened and fissured, but rarely an ulcerated, spot. There is pain and burning, which also causes a child to suck in air through the corners. There is no swelling of the lips in uncomplicated cases, nor any scarring afterward, though there may be some temporary discoloration.

DIAGNOSIS.—The disease sometimes superficially resembles herpes labialis and genuine impetigo, but is really liable to confusion with syphilis only, from which it is distinguished by the history, constitutional and other local signs of lues, as well as by the Wassermann test. Perlèche is confined to one region, while syphilis is not.

COMPLICATIONS.—There are no complications.

TREATMENT.—*Prophylactic.*—The disease is prevented through cleanliness and avoidance of contagion.

Curative.—Several methods of curative treatment are advised. **Nitrate of silver** (10 per cent. to 50 per cent.), **diluted tincture of iodin,** and **sulphate of copper** have their advocates. André claims a cure of a very obstinate case with 5 per cent. **chromic acid.** Dunn recommends **boric ointment** first to soften the scabs and then advises touching with **nitrate of silver,** which is to be followed by **boric ointment** continuously.

PROGNOSIS.—Most cases will heal without any treatment, but proper therapeutics hasten the cure.

Fordyce's Disease is characterized by minute white and yellowish nodules under the mucosa of the cheeks and lips. It never appears on the palate or tongue, but is very intractable.

Pyorrhœa Alveolaris

SYNONYMS.—Rigg's disease, periostitis alveolaris dentalis.

DEFINITION.—This is a destruction of the pericemental membrane and inflammation of the gums overlying the affected teeth. Riesman recognizes a local and a systemic form. This condition is almost universal after middle life.

ETIOLOGY.—Benedict and Riesman both consider heredity a causal factor, especially in the gouty systemic form. Carelessness and neglect of the teeth, trauma resulting from stiff brushes, toothpicks or threads,

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produce the atrium for infection for which the multitude of oral denizens are eagerly waiting.

While not rigidly specific, Ulrich calls these cases a "streptococcicosis." Both the viridans and hemolyticus, as well as the pneumococcus, are criminally concerned.

The entameba for which Bass claimed so much responsibility has been largely exonerated from blame. Williams suggested that it might be a beneficent scavenger, as he found it in 35 per cent. of mouths of healthy children from 5 to 7 years of age; but it increases in frequency and numbers in institutional subjects, especially if they have dirty teeth. Herbivorous animals are exempt.

Symptoms.—The mucous membrane of the gums becomes deeply congested and separates from the teeth when the pericemental membrane is destroyed. Pockets are thus formed between the gum and the denuded teeth, which may extend down into the alveolar process. These are filled with pus, food débris and saliva which can be expressed mechanically. The teeth are tender, and even without decay may loosen and fall out, owing to recession of the gums. This may become a chronic and intractable disorder and a source of systemic poisoning, as the inflammatory products, as well as the decaying food particles, are being constantly swallowed or absorbed locally. Bad breath is a characteristic symptom.

Diagnosis.—The condition is easily recognized on careful inspection, as several teeth are generally affected, most commonly the incisors first.

Osler's test—by which the condition is detected if any offensive odor clings to a thread drawn between the teeth at the gum margin—seems rather severe, as tenacious food remnants are often responsible for disagreeable olfactory appeals in this locality.

Association with Other Diseases.—Pyorrhœa alveolaris may be an early indication of diabetes mellitus and tabes dorsalis. Various sequelæ consequent upon resulting oral sepsis will be referred to under that subject.

Treatment.—Treatment is largely in the dentist's hands, as far as the local process is concerned, but there should be active and constant association with the internist, in order to obviate and relieve the possible systemic consequences. All salivary deposits should be removed and the teeth cleaned and polished. The gums should be treated with stimulating applications which possess germicidal powers, such as **tincture** of **iodin.** Though injections of **emetin** are no longer generally used, some **ipecac** may be added to mouth washes, in order to discourage the ever present entamebæ, despite the small, if any, part they are now credited with in the pathogenesis. **Optochin** has been recommended for pneumococcic cases.

Tilden claims that no pyorrhœa nor tooth caries will occur if the diet is free from salt meats and refined flours; i.e., if we keep our alkaline reserve. Howe emphasizes the importance of an ample and available supply of calcium.

"The Defensive Diet League of America" has attempted to popularize these notions.

Diet for pyorrhœa requires minimum of fats, sweets and heavy meats and maximum of fruits, grains and vegetables; i.e., alkaline dietary.

Prognosis.—Pyorrhœa alveolaris is a most obstinate and protracted trouble which requires the utmost care, persistence and thoroughness in order to effect eradication. Unfortunately, the condition is not always or commonly detected until it has gained a firm foothold, and it is then impregnably intrenched about many teeth.

PATHOLOGY.—There is, in the local form, a denuding of the dentine, where the enamel, the gum, epithelium, periodontium and cementum come together, by the deposition of salivary calculi. This is the only place in the body where bony tissues are unprotected. The systemic form may arise from vascular disorders in the pericemental membrane, following upon such constitutional diseases as nephritis, gout, tuberculosis, syphilis, diabetes mellitus or arteriosclerosis. The deposits here are too high up on the root to be seen, and are said to be derived from the blood, being urates in gouty cases.

Fetor Oris

SYNONYMS.—Bad breath, offensive breath.

ETIOLOGY.—The etiology is very varied. Some cases seem to be hereditary. The writer knows of a family in which bad breath has been manifested in three generations and in several members of each: a sort of ''heirloom'' handed down, which none of them perceive in each other.

It is observed in all forms of stomatitis, caries of the teeth, pyorrhœa alveolaris, local septic conditions, adenoids and enlarged tonsils, especially when the crypts hold cheese-like masses of epithelial débris, bacteria and food remnants, which on mechanical disintegration emit a fearful odor. Nasal catarrh and sinus troubles may be responsible. The source is sometimes further down in the digestive tract, such as esophageal diverticula, containing decomposing food, and all forms of gastric and intestinal catarrh. Rolleston says that cirrhosis of the liver may give a characteristic odor to the breath, which resembles dried and decomposing blood. Stagnation in the large bowel may facilitate the reabsorption of volatile fecal products, and the subsequent elimination of the latter through the respiratory passages may impart a distinctive smell. Diseases of the larynx, bronchi and lungs—such as ulceration, fetid bronchitis and gangrene—produce offensive breath.

Certain infections, such as diphtheria (chiefly from its local ravages) and typhoid, are attended by characteristic odors, and all febrile conditions impart a heavy smell to the respiration. Diabetes, especially with ketonuria, has the acetone or fruity breath, and the odor of ammonia may be due to uremia. The new-mown hay smell is strongly suggestive of pyemia. Tobacco and alcohol impart characteristic odors, as do also certain drugs, such as phosphorus, asafetida, bromids and iodids. Arsenic produces a smell like onions; this is sometimes useful as a therapeutic guide, when the drug is administered hypodermatically.

As Osler points out, the two most common causes are pyorrhœa alveolaris and chronic lacunar tonsillitis.

SYMPTOMS.—This condition involves both the taste and smell, and a breath which is disagreeable to one person may not be so to another.

The symptoms are chiefly objective, as in the most objectionable cases the patients are often totally unaware of their trouble.

TREATMENT.—The treatment is etiological, though **antiseptic and aromatic mouth washes and gargles** are helpful in disguising the worst features.

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PROGNOSIS.—The prognosis depends upon the ability to remove the cause, and this is often difficult, even when discoverable.

Oral Sepsis

ETIOLOGY.—The mouth is the largest and most patulous orifice of the body, and, moreover, the least able to defend itself against infection and abuse. Substances are put into it and kept there that no other part would tolerate. It opens up at birth and, in many cases, still unclosed, adds terror to the death agony. It early becomes infested with innumerable reputable and disreputable microörganisms, styled by Cigrand "the menagerie of the mouth," which may, in the careless or debilitated, establish themselves impregnably in the various recesses of the oral cavity. Consequently septic processes sooner or later arise, with disastrous consequences to the subject.

As to the *causation,* saliva and bacteria have their respective advocates as being the original offenders, but so far no satisfactory changes in the salivary content or reaction have been discovered, at least such as would render the teeth and gums peculiarly liable to infection. The affinity of various bacteria for certain tissues and the transmutation of some different forms are insisted upon by Rosenow. Acid fermentation of carbohydrates is accorded the chief rôle by Gies, who advises a return to the ancient Roman custom of closing meals with fruits, especially apples, which remove or neutralize the offending material. Brushing the teeth disposes of three-fourths of the bacteria and tobacco chewing may have some palliation and even virtue in the same direction.

DIAGNOSIS.—The diagnosis can only be made by very careful inspection, supplemented by *x*-rays, which should be taken by one well versed in this locality and skillful in interpretation. Shadows at the apices of teeth roots practically always mean infection, but sometimes a discharging sinus and even a granuloma does not cast a shadow. Devitalized and bridged teeth should always be considered with suspicion. No physical examination is complete without a careful study of the mouth. The teeth may have been blamed too much, but they will bear watching. Sometimes the mouth changes are secondary, but not as often as some seem to think.

ASSOCIATION WITH OTHER DISEASES.—Pyorrhœa alveolaris and bad breath, considered above, are closely associated with this condition, as well as alveolar abscess and various forms of ulceration. While William Hunter deserves the honor of calling scientific attention to the dangers of oral sepsis, the subject was suggested by Hippocrates, who thought constitutional effects might follow dental lesions, though he was mainly concerned with dentition. Benjamin Rush, one of the Signers of the Declaration of Independence, observed that rheumatism, dyspepsia and epilepsy were all relieved by the removal of offending teeth. Miller, of Berlin, also, deserves to be mentioned as a pioneer in this subject.

The teeth are the most frequent sources of trouble here, and we now recognize their enormous importance to health, as well as to our pulchritude and happiness. Woods Hutchinson says, "A man is known by the teeth he keeps." People with poor teeth are not apt to have good health, and many with poor health are apt to have poor teeth. There is very marked progress now being made in dental research and dental therapeutics, to the great and lasting benefit of the race. Efforts were formerly concentrated on keeping the teeth in the jaw for cosmetic, masticatory or musical reasons without much, if any, consideration of possible remote effects on the general system. Bridges were engineered, crowns conferred, and dead, infected roots filled, with resulting perennial gum boils. These foci need not be in any acute or obvious activity, but chronic processes, undetectable except by x-rays, may be responsible for the most disastrous outcome. The patients frequently do not notice the condition and are astonished when attention is called to it or when its connection with some systemic disease, for which relief is sought, is suggested. A realization of this danger now gives dentistry the added and higher aim of the prevention and cure of systemic diseases. It is a highly specialized branch of surgery.

According to Thoma, the *mode of distribution* is sixfold:

(1) By continuity. Necrosis of the maxilla, invasion of the antrum and sinuses may follow.

(2) Infection of the gastro-intestinal tract through the continual swallowing of pus is strongly presumed but not agreed to by all. Smithies has shown that bacteria are present in 87 per cent. even of acid stomachs; so that normal gastric juice may not be such a perfect antiseptic, and its feeble powers may be completely overcome by a persistent and unremitting onslaught from the mouth. Gastric and duodenal ulcers may thus be entailed, as well as appendicitis and colitis.

(3) Lymphogenous infection. Lymph-glands are often involved in the vicinity of gum boils and sore mouths, and then those of the neck may succumb. This condition is more frequent in children, and perhaps tuberculosis may thus gain access oftener than we think. Cantani has reported a number of such cases. Infection may follow the lymph-spaces of nerve-trunks and so reach the brain and cord.

(4) Hematogenous infection. Lesions without direct outlet, such as blind abscesses and periodontal cysts, are most liable to be a source of absorption into the blood. This is the most frequent method.

(5) Indirectly a chronic mouth infection may impose a great burden on the system weakened by some other disease. Thoma cites 2 cases of endocarditis which resisted every other measure until two unoffending teeth, shown by x-ray to be abscessed, were removed, and then cure promptly followed.

(6) A subjective sense of pressure and nervous and glandular disorders may be due to diseased teeth. Many neuralgias are dental in origin. The functions of the endocrinous glands—especially the thyroid, pancreas and adrenals—may be seriously disturbed by carious teeth, resulting in hyperthyroidism and diabetes. In general medicine, sepsis and pyemia may be due to decayed or abscessed teeth. Severe cases are, fortunately, rare, but there are a great number of vague medical conditions that are probably due to such causes and may eventuate into something more serious, in case the general resistance is lowered in any way. Some of these nondescript conditions may be seen "in disproportionate fatigue from slight exertion, inability to do a full day's work," benumbed mental activity, loss of weight, "irregular variations in temperature" sometimes called "febricula"—that opprobrium of diagnosis, which we always blush to inscribe. Leukocytosis and secondary anemias are often observed. Primary pernicious anemia was long ago attributed to mouth infection. Vascular hypertension, nephritis and even arteriosclerosis

may supervene, as well as many obstinate arthritides. Even virtuous genito-urinary troubles have been ascribed to gum boils. Furunculosis, also, has arisen from this cause, as well as erythema nodosum, erythema multiforme, and even scleroderma and Raynaud's disease. Iritis, keratitis and optic atrophy have been proved by Haden experimentally to be due to oral infection. Tooth infections are extremely frequent. Black reports 56 per cent. in persons under 25 years; 72 per cent. between 25 and 30; 87 per cent. between 30 and 40; 89 per cent. between 40 and 50; and 100 per cent. in all over 50 years.

Oral sepsis is then a source of ill health in many ways. It harbors and distributes disease germs. It may have a toxic effect, slight in the otherwise healthy, but disastrous to the debilitated, dissipated or aged. It may encourage other infections too weak or too modest to assert themselves independently. Duke thinks it may furnish the alien protein to which the patient is sensitized or may increase functional disturbance due primarily to independent organic disease, so that anaphylaxis shown in angioneurotic edema, nettle rash, etc., may follow. Not every one with oral sepsis has constitutional trouble, but it should always be corrected for preventive purposes.

Diagnosis.—Torrens claims that there is a specific type of leukocyte found only in oral infections. "A large lymphocyte, irregular in contour, whose cytoplasm has a grayish grainy appearance. The nucleus is irregular and stains with moderate density, frequently cuboid or ovoid with one flattened side." This is not due to pressure. The cytoplasm frequently shows vacuole and acidophil granulations. Technique must be perfect and lues and early tuberculosis distinguished by their history and other symptoms. Haden, however, denies this conclusion after much study.

Guthrie contends that a green stain extending up on the tooth is suggestive of pyelitis. It is associated with lack of vitamin C and is characterized by a distaste for acids, which cannot be distinguished from bitter substances.

Weber reports dark pigmented spots on the oral mucosa not connected with Addison's Disease, but with tuberculosis elsewhere and also in pernicious anæmia.

Haden found more bacteria at the roots of dead teeth whose *x*-rays were negative than in those that showed abscesses.

Treatment.—The treament should be carried out by the dentist and physician in cordial coöperation. Under these conditions the prognosis is often very good; but measures must be prompt.

The teeth are as important as the tonsils and should not be any more ruthlessly spared or sacrificed. Especially should milk teeth be preserved to their allotted span, that the jaw may develop and the permanent set mature. Vaccine therapy—even the autogenous—often disappoints. The author agrees with White in his claim that all infected teeth should not be removed at once, as the chance of making any more autogenous vaccine will then be lost, or too violent a reaction may thereby be stirred up in those already heavily infected. Curettement of the socket is often necessary. Emerson says a patient is rarely cured by attention to the teeth alone. The process has extended to other parts and must be relieved and broken up in them also, where, alas, too often it has become impregnably intrenched.

Early and thorough eradication of the focus then is essential, which may involve a real surgical operation. Mere extraction and blind curettement may really do harm by leaving behind some thereby invigorated infection as Burns and Black found out.

In chronic cases with leukopenia nuclein must be given to relieve the anaphylaxis that this is thought to indicate.

PATHOLOGY.—Impacted and unerupted teeth, obscure pericemental inflammation and affections of the dental pulp, such as irritation from improper filling and infection from obscure decay, with consequent pyorrhea and alveolar abscess, are the chief pathological conditions.

Of the innumerable bacteria present, Thoma enumerates 16 pathogenic species, 5 of which are strict anaërobes and dangerous denizens. The *Streptococcus hemolyticus* in acute, and the *Streptococcus viridans* in chronic abscesses, are the most common. Pneumococcus and gonococcus are less frequently seen. In carious teeth or in the roots is the best possible place for these germs to find lodgment and remain undisturbed. Safe there from our natural defenses, they acquire high resistance, by being kept irritated and exercised by the pressure of mastication. The socket is a true gomphosis joint and especially favors infection, because it has no capsule and an abundant blood supply. Dental decay in children is often due to oral ingestion of carbohydrates, especially holding candy in the mouth. Repeated stomatitis, as stated, is influential in the same direction. Soft foods make soft teeth.

Leukoplakia Buccalis

SYNONYMS.—Leukoplasia oris, psoriasis linguæ, ichthyosis buccalis, smoker's tongue.

ETIOLOGY.—Adult males who smoke are the chief victims. It is rare in women, even when they use tobacco, and unknown in children, though Montgomery reports a case in a luetic boy of sixteen, who had no other small vices.

Heat from tobacco smoke or glass-blowing seems to cause this condition, and other local irritants such as broken teeth, irritating food and alcohol, are occasionally responsible. Syphilis has probably been unjustly accused, but it certainly does not confer immunity.

SYMPTOMS.—This is a chronic affection of the mucous membrane of the mouth, especially of the tongue, characterized by the development of whitish or yellowish slightly elevated and indurated patches, which are either fissured or smooth.

The symptoms are not prominent subjectively, though a vague sense of discomfort and perversion of sensation may be complained of. The subject unconsciously rubs his tongue over the affected areas and so keeps them irritated, though no pain is felt; but there is a not unfounded apprehension of malignancy which depresses the spirits. At first there are deep red patches, oftenest on the tongue, but soon invading the lips, gums, cheeks and palate, which thicken up and turn bluish-white. Warty excrescences may develop, which increase the danger of epithelioma.

DIAGNOSIS.—The disease has to be distinguished from syphilis, which is more apt to ulcerate and has corroborative lesions elsewhere.

TREATMENT.—The treatment consists in the **removal of all sources of irritation** and **prompt surgical intervention,** on the slightest threat

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of warty development or ulceration. **Radium** and **salvarsan** are sometimes useful.

Prognosis.—The condition is chronic and very obstinate, though, according to Osler, recovery has been observed. Carcinoma follows in about 20 per cent. of cases.

Pathology.—Pathologically, the process is neither psoriasis nor ichthyosis, but a hyperkeratosis of the horny layer of the mucous membrane. There is excessive activity of the deeper layers of the epidermis with infiltration of the congested papillary processes with leukocytes.

Ageusia

Ageusia, or loss of taste, may occur temporarily through local conditions, such as dryness from mouth breathing. It is then general. It is partial and may be more permanent in affections of the chorda tympani nerve, particularly in certain forms of facial paralysis, where the lesion is located in the petrous bone. Ageusia from glossopharyngeal involvement is quite unusual. Anesthesia of the mouth in fifth nerve lesions impairs taste, and all forms of stomatitis are attended by varying degrees of disturbance.

Hyperageusia

Hyperageusia is hysterical. Epileptic auræ occasionally are gustatory and suggest a lesion in the uncinate gyrus.

Parageusia

Perversion of taste, or parageusia, is often seen in hysteria and, regularly, in febrile cases as well as in most chronic diseases, where it is one of the main causes of anorexia. Reisman refers to a case, nondiabetic, of neurasthenia and moderate arteriosclerosis which had a constant sweet taste. Many uremics complain of a fearful taste resembling carrion, and the "dark brown" oral sensation of the "morning after" is familiar to the dissipated. The fetor oris of chronic constipation, referred to above, is sometimes perceptible to taste, especially on waking. In two otherwise obscure cases of the author's, inadequate renal function was finally found to be responsible, though no albumin or casts were ever discovered in the urine. Injection of phenolsulphophthalein showed where the trouble was.

There are still some cases, in which an ineradicable and inexplicable bad taste exists, that defy all efforts at investigation and relief. Such patients should be carefully watched, as the symptom may be a premonitory hallucination of insanity.

Geographical Tongue (*Eczema of the Tongue*)

This is not strictly a disease, but a propensity in young children especially, though shared with adults, to an irregular desquamation of the epithelium of the tongue, leading to denuded patches of varying and fantastic shapes. It begins as elevated, flattened, whitish spots on the dorsum of the tongue, which may come and go suddenly or change their shapes by fusion or extension. Pepper associated this condition with morbid cravings of the appetite, and it is commonly regarded as connected with chronic gastric disorders, though it certainly often persists after their apparent relief. It may be hereditary.

Pathologically it is an irregular heaping up of poorly formed epithelial cells. The causes are unknown. Speculation has been rife, but vain. Heredity and gout and even syphilis have been accused. A trophoneurosis has been suggested. It is certainly most often associated with gastro-intestinal disturbances, but no specific parasitic cause has been discovered.

DIAGNOSIS.—This condition cannot be confounded with any other. The tongue is no longer the diagnostic criterion it once was and its coatings retain, chiefly, pathological interest. Any one on a liquid diet, especially milk, will have a white, furry tongue, because mastication becomes unnecessary. Both clean and coated tongues are found in digestive disorders. Typhoid and scarlet fever cling most obstinately to their special lingual appearances but not always successfully. A dry, beefy tongue, in asthenic cases, is more ominous than a moist, coated one. Any of these may temporarily assume the geographical appearance.

TREATMENT.—No treatment is necessary, and would be futile, the condition being harmless. It disappears spontaneously, but may return several times.

Nigrities (*Hairy Tongue, Black Tongue*)

This condition is scarcely more than a pathological curiosity. It consists in the great elongation and cornification of the epithelium of the filiform papillæ which become discolored. It begins on the center of the dorsum of the tongue and spreads to the edges, though the hue gets lighter. It is obstinate and may recur after removal by **scraping, or by frequent applications of peroxid of hydrogen or solutions of borax.**

It is seen in dyspeptics, syphilitics and hypochondriacs, and may be due to some obscure defect in nutrition or the ravages of some undiscovered parasite. No age is exempt, though it is a very rare affection. *So-called black tongue* may also be due to the growth of mucor.

White tongue, associated with a very bad taste, is more apt to be unilateral. The epithelium and papillæ become white.

Glossitis

ETIOLOGY.—The most frequent causes of glossitis, or inflammation of the tongue, are the bites of insects, burns, the tearing of the organ in an epileptic convulsion, or on a rough tooth. Tobacco, too, is sometimes suspected. Of the infections responsible for this condition, small-pox, erysipelas and scarlet fever are mentioned, but generally a severe stomatitis has preceded. Chronic glossitis may be due to streptococcus, saccharomyces and Friedlander's group.

SYMPTOMS.—Glossitis, as distinguished from other inflammatory conditions of the mouth, is usually traumatic or associated with some infectious disease. It is marked by great swelling, so that the organ may protrude from the mouth and interfere seriously with the taking of nourishment, phonation and even respiration, by pressing upon the epiglottis and soft palate. If traumatic in origin, the point of injury may be covered with a necrotic membrane. Fever, pain and enlargement of lingual lymphatics is common, and abscess may develop. A chronic form sometimes followed by atrophy has been described. So-called Moeller's Glossitis is characterized by abnormal smoothness of the tongue's

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surface and a loss of papillæ with a tendency to ulcer formation. Acids are especially painful.

TREATMENT.—The condition is unmistakable and is treated by **cold and demulcent applications,** with an **early resort to surgery,** if pus can be located or tension becomes extreme. Olive oil is the best application and vehicle for sore tongue.

PROGNOSIS.—The prognosis is generally good, though a fatal issue in forty-eight hours is not unknown.

Ulcers of the tongue are traumatic, tuberculous, syphilitic and neoplastic. They are thus described by Handfrield Jones. The traumatic are of varied depth, a pinkish yellow color and apt to be painful. The edges are indurated and sloping.

The tuberculous form is shallow, grayish yellow and also painful. The edges are sinuous and not undermined, but sloping.

The syphilitic and neoplastic are both deep and gray in color and not painful, locally, though there is often referred suffering in the malignant forms. The edges of the luetic form are punched out, ragged and undermined, while the neoplastic are raised, everted and ragged.

Treatment is etiologic. The prognosis is good except in the tuberculous and malignant forms.

Blastomycosis

Blastomycosis of the tongue is very rare. It presents itself as a warty tumor at the base, and may extend to the larynx. New claims its "ready relief" by **iodin locally** and **potassium iodid internally. Radium,** too, has been advised.

Glossodynia

Glossodynia, or neuralgia of the tongue, may be due to lingual tonsillitis, to which local therapeutic efforts must be accordingly directed.

The congenital midline protrusion of the hard palate has been designated *Torus Palatinus.* It is more frequent in females and thought by some to be a sign of tuberculosis, syphilis, pulmonary osteo-arthropathy or degeneracy, though no conclusive evidence therefor has been adduced.

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CHAPTER III

DISEASES OF THE PANCREAS

By Henry Joachim, M.D.

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Special diseases of the pancreas, p. 35:—Diseases of the pancreatic ducts, p. 35:—Anomalies of the ducts, p. 35; Inflammations of the pancreatic ducts (sialo-angitis, sialodochitis), p. 36; Pancreatic calculi, or sialolithiasis, p. 38; Pancreatic cysts, p. 40—Diseases of the pancreatic blood-vessels, p. 45—Diseases of the pancreatic parenchyma, p. 46; Acute hemorrhagic pancreatitis, p. 46; Pancreatic abscess, p. 52; Fat necrosis, p. 52; Tumors of the pancreas, p. 53—Diseases of the stroma, p. 55—Chronic interstitial pancreatitis, p. 55—Chronic interlobular pancreatitis, p. 56; Interacinar pancreatitis, p. 58.

GENERAL PATHOLOGY

A systematic clinical study of diseases of the pancreas was begun about twenty-five years ago. Prior to this our clinical knowledge of this organ was rather fragmentary and rudimentary, and was incommensurate with its physiological importance. Isolated pathological data were limited to accidental postmortem or operative findings. Physically inaccessible to direct examination on account of its deep anatomical position and environment, functionally interdependent upon gastric, hepatic and duodenal digestion, it eluded the skill of the older clinicians as a factor in the production of disease.

Physiologically, the pancreas has two distinct functions: one, the internal or metabolic function, concerned mostly with sugar metabolism (*see* Diabetes, Vol. IX, Chapter VI, pp. 69-134) and growth and dependent upon the integrity of the islands of Langerhans; the other, the external or alimentary function. This article confines itself entirely to the pathology and symptomatology of the latter.

Since the diagnosis of pancreatic diseases depends almost entirely upon the study of the physiological deficiencies and aberrations of this organ, or of the changes produced in adjacent organs and structures, a brief practical survey of the chief anatomical and physiological data will be considered. From a diagnostic standpoint the following anatomical relationships are of importance:

(1) The pancreatic head is almost completely surrounded by the duodenum.

(2) The anterior surface is separated from the stomach and colon by the lesser peritoneal sac. A small part of the general peritoneal cavity impinges against the antero-inferior angle.

(3) Closely hugging the head of the pancreas and often imbedded in it is the common bile-duct.

(4) Posteriorly, the following structures have some significance in pancreatic pathology: the aorta, inferior vena cava, mesenteric, portal, renal, and splenic vessels, the receptaculum chyli and crura of the diaphragm. The spleen, left kidney and suprarenal body are in contact with the tail of the pancreas.

(5) Inferiorly, the jejunum is in relation to the body of the pancreas.

(6) The practical anatomy of the ducts will be considered in the discussion of their anomalies.

Physiologically, as demonstrated by Pawlow, the pancreatic juice is secreted during gastric digestion and not during the intervals. The gastric juice must, in addition, contain hydrochloric acid. This is the normal stimulus to the secretion of the pancreatic juice, since by its action on the duodenal mucosa it generates a substance called secretin, which is the pancreatic hormone (Bayliss and Starling). The juice which is secreted possesses no digestive properties while in contact with the pancreatic alveoli and ducts. Normally it becomes activated only upon its arrival in the duodenum. Here it comes into contact with the enterokinase, which is secreted by the mucosa of the small intestine. This, by its action on the inactive trypsinogen, converts the latter into the ferment trypsin. In the duodenum the bile acts as the activator of the pancreatic lipase. Under ordinary circumstances neither bile nor enterokinase enter the pancreatic ducts. It is said that calcium salts possess activating properties for both the proteolytic and lipolytic ferments. As far as is known, the starch-splitting ferment needs no activation. Concerning the milk-curdling ferment, little more is known than that it acts on unboiled milk and not on boiled milk (Einhorn).

This leads up to the question so important in pancreatic diseases: Why does not the pancreas normally digest itself? The physiological principle enunciated in the preceding paragraph offers a satisfactory explanation. If under any circumstances duodenal contents find their way into the ducts or alveoli, the factors which produce autolysis or self-digestion become potent. The duodenum is safeguarded by the rapid combination of the pancreatic juice with the acid chyme, by the lability and ease of destruction of the excess ferment by intestinal bacteria, or perhaps by the presence of an antitryptic ferment in the blood or intestinal mucosa which neutralizes its action. This antitryptic substance is highly developed in the *Ascaris lumbricoides* (round worm), which thrives in the duodenum, and explains its immunity to pancreatic digestion.

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GENERAL DIAGNOSIS

The pancreas may be the seat of gross and extensive pathology without there being any clinical manifestations during life, and with little or no functional disturbance. This is explained by the probable compensatory action of the other digestive and bacterial (intestinal) fluids. Negative clinical or laboratory findings do not therefore exclude the possibility of organic structural changes in the pancreas.

In the diagnosis of pancreatic insufficiency we study its effect on the digestive functions and on sugar metabolism. Normally the pancreas digests proteins to the stage of amino-acids. It is the only digestive fluid which acts upon the nucleins (cell-nuclei) and changes neutral fats into fatty acids, soaps and glycerin. It acts upon both boiled and unboiled starches. Interference with any or all of these functions results in deficiency of the digestion of these substances.

In general we have three methods of investigation:

(1) Examination of the feces for the presence or absence of the products of pancreatic digestion.

(2) Duodenal intubation according to the method of Einhorn, which permits a direct study of the pancreatic juice as it is secreted.

(3) Internal secretory activity as determined by a study of the sugar tolerance and the Loewi adrenalin test.

Evidence of Pancreatic Disease as Determined from the Feces.—For general diagnostic purposes a modified Schmidt intestinal test-diet (*see* Diseases of the Intestines, Vol. VII, Chapter XI) may be taken. It consists of some milk and cocoa, two slices of bread and butter, or toast, rice broth, rare steak or chopped beef, potato, and a dessert. Carmin in capsules, or charcoal tablets, may be used for stool demarcation. Grossly, the pancreatic stool is bulky, white, clay-colored or asbestos-like in appearance, exceedingly offensive, and coated with a floating, oily layer. Acholic stools due to biliary obstruction present much the same appearance and cannot be differentiated by a gross examination. Often both conditions coëxist.

A small specimen of the feces is placed upon a microscopical slide to which a drop of Lugol's solution has been added. The specimen and reagent are thoroughly mixed upon the slide with a glass stirring rod and examined under a cover glass (air-bubbles should be excluded) with the diaphragm shut down and with a 1/6 lens.

Azotorrhea.—On glancing at the specimen, one may be impressed with the large number of transversely striated muscle-fibers with well-preserved nuclei under the sarcolemma. To this condition the term azotorrhea (literally a flow of nitrogen) has been applied. A better and more suggestive name is *creatorrhea.* One must exercise a little caution before drawing a conclusion of pancreatic insufficiency from the presence of this manifestation without further corroborative evidence. In achylia gastrica the connective tissue surrounding the muscle-fiber remains intact, and therefore when the meat-fiber encased in this connective tissue capsule reaches the duodenum, it is protected from the action of the pancreatic juice. In this condition one can to some extent overcome this gastric creatorrhea by cutting the meat transversely to the direction of the muscle-fiber, before ingestion, thereby opening up the intact connective tissue envelop and exposing the fibers to tryptic action. On the other hand, the pancreatic juice is deficient in cases of achylia due to the absence of the hormone excitor, i. e., the hydrochloric acid. The author has often seen the creatorrhea and diarrhea in these cases yield to large doses of hydrochloric acid.

Another factor in the presence of creatorrhea is a hyperperistalsis (catharsis) or the presence of a gastrocolic fistula (lientery). Neither of these conditions permits intimate or sufficient contact for the proper completion of pancreatic digestion. Excessive indulgence in meats sometimes results in an excretion of undigested fibers. To recapitulate, creatorrhea signifies:

(1) Excessive meat intake.
(2) Faulty gastric digestion—achylia gastrica.
(3) Hyperperistalsis from catharsis, enteritis, etc.
(4) Gastrocolic fistula (carcinoma, ulcer).
(5) Pancreatic insufficiency.

There are two other tests which may be resorted to in the demonstration of tryptic deficiency. They are somewhat more complicated and time-consuming and are no more conclusive than the simple microscopical examination. They are:

Sahli Glutoid Test.—Weyland observed that gelatin hardened in formalin (glutoid) escaped the action of the gastric juice but not that of the pancreas. He applied this therapeutically by prescribing drugs that acted as gastric irritants in these capsules. Sahli conceived the idea that this method could be utilized in the diagnosis of tryptic insufficiency. He administered substances in these capsules, which are quickly broken down and absorbed in the duodenum as soon as they are liberated and are rapidly eliminated in the urine or in the saliva. Either iodoform, potassium iodid, or salol may be used. Iodin may be detected in the saliva or urine within a period of two hours when iodids are utilized in performing the test, or salicyluric acid in the urine when salol is used. A simple method which the author has used for the detection of iodids in the saliva consists in placing a calomel tablet upon or under the tongue, which in the presence of iodin turns to a yellow color by the formation of the yellow iodid of mercury. Recently the author has found that calomel tablets were made up with starch. Instead of the yellow color a blue color was formed (iodin and starch reaction). When salol is used for the test, a blue color is imparted to the urine on the addition of a solution of ferric chlorid. One of the objections advanced to the Sahli test is the fact that gastric motility enters as an

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element; but Sahli eliminates this factor by previously testing the motor activity of the stomach. Delayed gastric motility gives delayed or absent salivary or urinary reactions.

Schmidt's Nuclein Test.—This test depends upon the fact that cell-nuclei are digested by the pancreatic juice (nuclease ?). Small cubes of beef, hardened in alcohol and washed before performing the test, are placed in a small gauze or silk bag and ingested with a meal. The bag is recovered from the feces and the contents are examined microscopically after the application of nuclear stains. The absence of any nuclei is considered evidence of good pancreatic digestion, provided the bag has not been too long delayed in its passage through the intestinal tract. If exposed too long to the action of the intestinal bacteria, the latter decompose nucleins, and a putrefactive karyolysis results in the absence of pancreatic digestion. Diarrhea prevents nuclear digestion (rapid peristalsis). Kashiwado has modified this test by using the thymus gland (neck sweet-bread), which is rich in nuclei, instead of the beef cubes. For details of these tests, reference may be made to special works on the feces. Trypsin can also be directly demonstrated in the feces. The tryptic action of leukocytes must be excluded.

STEATORRHEA.—By this term is understood the presence of an excessive amount of neutral fat. In the examination of the microscopical specimen a great many globular, highly refractile masses are found. Normally about 8 per cent. of fat is passed unchanged in the feces as neutral fat; the remainder is converted into fatty acid crystals which combine with calcium and magnesium to form soaps. In deficiency of lipolytic action almost all the neutral fat except milk fat is passed as such in the feces. It is quite unnecessary to use stains for demonstrating the fat. The needle-shaped fatty acid crystals and the yellow waxy calcium soaps are absent. This is of some importance in differentiating the steatorrhea of acholia, in which there is little interference with fat splitting, but only with fat absorption. In the latter condition, both fatty acid crystals and soaps are abundantly found.

It may seem superfluous to say that air-bubbles must not be mistaken for fat, but, on more than one occasion, the author has seen this mistake made. In all, there are ten differential possibilities which present themselves in the diagnosis of steatorrhea:

1. Excessive intake of fats or oils (olive and cod-liver) in the treatment of inanition and tuberculosis.
2. Absence of bile in the intestines—acholia.
3. Diminished absorption of fat from extensive intestinal ulceration, acholia, amyloid or tuberculous mesenteric disease (tabes mesenterica), and from obstruction or disease of the thoracic duct.
4. Hyperperistalsis from catharsis, enteritis, and gastrocolic fistula.
5. The ingestion of indigestible oils, as castor or paraffin oils, such as are now in vogue in the treatment of constipation.

6. The use of suppositories containing an oily base (theobromin) or glycerin. Patients often bring a specimen of feces for examination, which is procured by means of suppositories.
7. Psilosis or tropical sprue, which is probably associated with pancreatic insufficiency.
8. Pellagra.
9. The diarrheal crises of hyperthyroidism. In this disease there may be an inhibition of the pancreas.
10. Pancreatic disease.

Winternitz has introduced an ingenious method for the determination of the lipolytic action of the pancreas. It depends upon the fact that iodin in oily combination, as in iodipin and sajodin, is not liberated except in the presence of the fat-splitting pancreatic ferment. Iodin is then demonstrated in the saliva or urine in the usual manner. The author has often failed to find iodin in the saliva of patients with essential hypertension who were taking sajodin. In some of these cases he has been able to demonstrate evidences of faulty pancreatic digestion in the feces. Some of these cases improved subjectively on pancreatic and intestinal treatment. The presence of excess of starch (amylorrhea) is not of diagnostic significance.

The Examination of the Duodenal Contents.—The presence of oil in the stomach causes a regurgitation of the duodenal contents. Volhard has utilized this by removing the oil and duodenal contents and then isolating the former. The pure duodenal juice mixed with bile was then examined. This method has been entirely superseded by the procedure introduced by Dr. Max Einhorn and known by the name of duodenal intubation. The apparatus consists of a perforated metallic capsule attached to fine rubber tubing, to the other end of which a glass syringe is attached. The capsule is then swallowed with a little water and at the end of five or six hours has usually reached the duodenum. The pancreatic juice, mixed with the bile, can then be aspirated. Rehfuss has somewhat improved the capsule by substituting longitudinal slits for the small perforations of Einhorn, which occasionally became clogged. For the technic of the introduction of the tube, reference must be made to Einhorn's original articles (*see* bibliography at end of article). The aspirated duodenal contents are then examined for the presence or absence of the pancreatic ferments. The absence of one ferment, as determined by examination, does not necessarily imply the absence of the others.

Tests for Disturbed Internal Secretion.—These tests include the study of sugar tolerance as described in the article on Diabetes, and the pupillary adrenalin test of Loewi. In pancreatic disease the inhibitory action of the pancreas is released from the antagonistic suprarenal gland, which responds by its dilating action on the pupil when a drop of a solution of 1-1000 adrenalin chlorid is instilled into the conjunctival sac (sympathetic stimulation). This test has proved unreliable in the author's experience.

Summarizing the tests, we have:

1. The macroscopic characteristics of the feces.
2. The presence of creatorrhea.
3. Schmidt's nuclein test and the Kashiwado thymus modification.
4. Sahli's glutoid test.
5. Steatorrhea.
6. Winternitz's sajodin test.
7. The duodenal intubation method of Einhorn.
8. Chemical and biological test in the feces. (For further details *see* tests in physiological chemistries.)

SPECIAL DISEASES OF THE PANCREAS

For convenience of clinical description the special diseases of the pancreas will be considered from the standpoint of:

1. Excretory ducts.
2. Blood-vessels.
3. Parenchyma.
4. Stroma.

No sharp lines of demarcation can be drawn in this classification, since seldom is one of these structures involved without some implication of the others. Therefore, it can hardly be assumed that each structure produces a separate clinical pathognomonic syndrome.

Diseases of the Pancreatic Ducts

These diseases will be discussed under the following heads:

1. Anomalies of the ducts.
2. Inflammations—Sialo-angitis.
3. Calculi—Sialolithiasis.
4. Cysts.

Anomalies of the Ducts.—Embryologically the pancreas is developed from three separate outgrowths which spring bud-like from the duodenum: one posterior sprout which later develops into the head, and two ventral buds which form the body and tail. The posterior segment has a duct which empties into the duodenum (Santorini's duct), and the two anterior segments empty by means of a larger duct (Wirsung's duct). Later, as development proceeds, the three segments fuse. The ducts usually retain their identity as they enter the duodenum, but communicate at an acute angle within the substance of the pancreas. Wirsung's duct opens in conjunction with the common bile-duct into the ampulla of Vater about 10 cm. from the pyloric orifice, and the other duct about 8 cm. from the latter. That portion of pancreatic tissue enclosed within the acute angle formed by the ducts is known as Desjardin's triangle. The arrangement, perviousness, and anomalies of the ducts are of great significance in the pathology of pancreatic diseases, from the standpoint of drainage and infection.

The chief variations studied by Opie, who has done most of the work on this phase of pancreatic pathology, are:

(1) The duct of Santorini may be impervious or absent. This implies that the whole drainage of the pancreatic juice depends upon the larger duct. There is no opportunity for compensatory or collateral drainage in the advent of obstruction of Wirsung's duct.

(2) Wirsung's duct may join the common bile-duct some distance before it enters the duodenum.

(3) The common duct may open by a distinct orifice some distance from the pancreatic duct.

(4) Santorini's duct may open into the duodenum at the papilla of Vater in conjunction with the common duct. In that case it usually becomes the main duct of the pancreas, and Wirsung's duct the smaller. Occasionally the latter is impervious.

The significance of these various arrangements of ducts will be discussed when the question of pancreatic drainage is considered.

Aberrant or accessory pancreatic tissue is of no clinical interest, but annular pancreatic tissue surrounding the duodenum is of some importance because of the possibility of compression of the latter in enlargements or inflammations of the pancreas. Congenital deficiency has a relationship to a type of infantilism described by Byron Bramwell.

Inflammations of the Pancreatic Ducts (Sialo-angitis, Sialodochitis).—ETIOLOGY.—It is important to notice that the pancreatic duct differs from all other ducts in the fact that throughout practically its entire course it is imbedded within the pancreatic tissue (intrapancreatic). It is scarcely conceivable how the surrounding parenchyma can escape involvement by contiguity. Another factor to be noticed is the almost complete absence of unstriped muscular fibers in the duct and pancreas. Due to this circumstance, there is an absence of propelling force. Add to this the fact that the pancreatic juice is secreted at a very low pressure, and we have all the requirements which favor stasis and its consequences.

An inflammatory condition of the ducts is usually an ascending one from the duodenum (enterogenous) resulting from alcohol, ptomains, etc., or originating in the biliary passages (cholangitis and cholelithiasis). It may be toxic or infective (typhoid, mumps).

DIAGNOSIS.—The symptoms are usually obscured by the pre-existing gastroduodenitis (excessive vomiting, diarrhea), or by cholangitis (jaundice), or cholelithiasis. If the jaundice persists, if on duodenal intubation there are no evidences of pancreatic ferments in the duodenal contents, and if in addition there are evidences in the feces of pancreatic insufficiency, the diagnosis of sialo-angitis may be ventured.

Complications.—The disease may go on to complete resolution, or one of the following complications may arise:

(1) Acute hemorrhagic pancreatitis and fat necrosis may take place. The occurrence of this complication depends upon the arrangement of the excretory ducts. If the duct of Santorini is impervious, then no provision for drainage exists when the lumen of the only other duct becomes occluded. In consequence, the pancreatic juice is dammed and enters the lymphatics and peri-acinar tissues, causing autolysis and infection (suppurative and gangrenous pancreatitis).

(2) A low grade infective sialo-angitis is a predisposing factor to an interstitial fibrosis (diabetes) or to calculus formation.

(3) Pancreatic cysts may be formed.

Treatment.—*Dietetic.*—Force **fluids**, restrict **animal fats** with the exception of milk fat, and **limit** the intake of **proteins.**

Medicinal.—This mode of treatment is usually directed toward the primary condition, which is either a cholangitis (cholelithiasis), or a gastroduodenitis. The goal in treatment is the production of duodenal and biliary peristalsis, which aids the removal of the inflammatory products from the ducts and establishes adequate **drainage.** This also combats the infection and militates against the possibility of serious pancreatic suppuration. One of the best means of attaining all these results is by the method of **duodenal lavage** (Einhorn or Rehfuss) with a 25 per cent. **magnesium sulphate solution** as practiced by Lyons. As demonstrated by Meltzer, this procedure relaxed the circular muscular fibers surrounding the papilla of Vater (Oddi's sphincter) which, in the presence of inflammation, is in a spastic condition. Drainage is thereby facilitated for both the biliary and the pancreatic passages. Duodenal peristalsis also exerts an aspiratory action upon the ducts. **Carlsbad salt** (essentially a solution of sodium sulphate, sodium chlorid, and sodium bicarbonate) has also been used on account of its solvent action on mucus and antispasmodic effect on Oddi's sphincter. **Atropin** may be of some benefit. Experimentally, two drugs are found to be excreted by the pancreatic juice, salicylates and urotropin (helmitol). The administration of **pilocarpin,** which tends to increase the flow of pancreatic juice, is **contra-indicated,** as it has been experimentally demonstrated to produce fat necrosis and hemorrhage in the presence of an insurmountable obstruction of the ducts.

Surgical.—If jaundice persists and the evidences of pancreatic insufficiency become more apparent, it is justifiable to drain the biliary passages surgically in order to prevent fatal or serious complications.

Prognosis.—This depends entirely upon the presence of complications and upon the etiological factors.

Pathology.—There has never been adequate opportunity for the study and observation of uncomplicated cases of catarrhal inflammations of the ducts. Only in the presence of calculi or of a complicating hemorrhage has this study been possible. In these cases there is either a desquamation of the lining cells of the ducts, or an autolysis. The condition may be followed by suppuration and hemorrhage. There may be a periductal lymphocytic infiltration, with newly-formed connective tissue. This process is essentially analogous to that which occurs in the liver in biliary cirrhosis. The head of the pancreas may be swollen from edema or induration from the absence of drainage. If the smaller duct is impervious and the pancreatic stasis is complete, there may be a cell atrophy of the alveoli and cicatricial changes between the alveoli.

Pancreatic Calculi or Sialolithiasis.—ETIOLOGY.—This condition seldom rises above the *niveau* of a diagnostic suspicion. It usually results from a catarrhal condition of the ducts (stasis) or from an infection arising from the duodenum or bile-passages. Occasionally a biliary calculus may enter the pancreatic duct, particularly when both the bile and pancreatic ducts join before emptying into the duodenum. Conversely, a pancreatic calculus may become lodged in the ampulla, become bile-tinged, and be mistaken for a biliary calculus. Pancreatic calculi are sometimes bile-stained.

SYMPTOMATOLOGY.—Pain is usually the most prominent and agonizing symptom. It may resemble that of cholelithiasis. It is usually situated in the epigastrium, is stabbing or spasmodic in nature, and may radiate along the left costal margin to the left shoulder-blade or to the left of the spine. It abates little or not at all on the administration of large doses of morphin, thereby differing somewhat from the pain of gall-stones. The pain is probably caused by distension, inflammation of the ducts, and pressure on nerve-ganglia, while that of biliary colic is due mostly to muscle-spasm, which yields to opium and atropin. The pain sometimes begins after a hearty meal, as a result of the increased tension produced by the alimentary pancreatic secretion. Salivation may accompany the paroxysm. Sometimes there are evidences of shock, collapse, and cardiac insufficiency. When jaundice occurs it is due either to an associated biliary calculus, or to compression of the common duct by the pancreatic stone, or the enlarged and edematous head of the pancreas. Sometimes an impacted calculus at the ampulla will obstruct the common duct. There may be evidences of pancreatic insufficiency in the feces or, in rare cases, a calcium carbonate calculus may be recovered after a painstaking examination. If, in addition to the above symptoms, glycosuria, oxaluria, or bronzing of the skin are present, the diagnosis of pancreatic stone becomes probable. *X*-ray examinations have thus far not definitely detected pancreatic calculi.

DIAGNOSIS.—Lazarus says, "the presence of 'gall-stone colic' without icterus, occurring in men, in the presence of azotorrhea, steatorrhea, and glycosuria with the absence of gall-bladder enlargement and tenderness, should arouse the suspicion of a pancreatic calculus." From a differential standpoint it is necessary to consider:

1. Pylorospasm, which is usually relieved by heat, atropin, cocain, etc.

2. Cholelithiasis, distinguished by bile-pigment in the urine, tenderness over Robson's point, a palpable or tender gall-bladder, a tender and spastic trapezius muscle on the right side, and a tender point to the right of the tenth dorsal vertebra (right Boas point). Gall-stones and pancreatic stones may coëxist.

3. Left-sided renal colic, tenderness of the testicle, downward radiation of the pain, vesical irritability, hematuria and oxaluria. The latter may also be well marked in uncomplicated pancreatic disease.

4. Intestinal colic, or obstruction (Treitz's fossa), lead-poisoning (Burton's line). Sometimes a pancreatic calculus produces a paralytic ileus.

5. Inflammation of an undescended or retrocolic appendix.

6. Mesenteric arteriosclerotic, vasomotor, and anginal crises—relief may be obtained by the administration of nitrites. Usually this is accompanied by an elevation of blood pressure. In pancreatic calculus this is usually depressed.

7. Thrombosis of the mesenteric vessels—melena.

8. Posterior perforation of a gastric or duodenal ulcer.

9. Crises of tabes and angioneurotic edema.

10. Angina pectoris.

The presence of a concretion in the feces offers the following possibilities:

1. Enterolith and intestinal sand, usually consisting of ammonium magnesium phosphate or oxalate.

2. Calcific incrustations or ligneous plant residues.

3. Calcium soaps from the excessive ingestion of olive oil in the treatment of gall-stones (calcium oleate concretions).

4. Bismuth concretions after excessive or continued ingestion of this substance (revealed by x-ray examinations).

5. Gall-stones—usually cholesterin, or calcium bilirubin, or verdin.

6. Pancreatic calculus—calcium carbonate or phosphate.

Complications.—The conditions which may complicate the disease are:

1. Pancreatic hemorrhage, fat necrosis, and pancreatitis.
2. Pancreatic abscess.
3. Perforation of the stone into other structures (stomach, colon).
4. Pancreatic fibrosis with diabetes.
5. Cysts.
6. Obstruction of the common bile-duct.
7. Carcinoma of the head of the pancreas.

Treatment.—Treatment is directed toward the relief of the paroxysm, the removal or solution of the stone, prevention of further formation, and the treatment of the complications. During the acute attack it may be necessary to resort to the use of chloroform inhalations if the pain does not yield to narcotics. It may be necessary to treat shock and cardiac weakness by means of the usual remedies. There are three pos-

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sible means by which calculi may be removed: first, by solution; secondly, by increasing the flow of the pancreatic juice to mechanically dislodge the stone; and lastly, by surgery. It is doubtful whether any chemical agents could exert a solvent action; hydrochloric acid has been recommended. To increase the flow of pancreatic juice, frequent feedings and the administration of **hydrochloric acid, pilocarpin hydrochlorid,** 1/6 grain (0.01 gram), **citric, and lactic acid** have all been experimentally used. **Calomel,** judging by its action on the salivary glands (ptyalism), should exert an analogous effect upon the pancreas. Any agent which produces duodenal peristalsis might be of some benefit. **Surgery** offers the only sure method.

To prevent the formation of calculi, it is necessary to treat the sialo-angitis and biliary infection. Baldwin thinks that the tendency to lithiasis (calcium oxalate) can be controlled by a **diet** free from calcium oxalates. Such a diet consists of meat, citrated milk, eggs, sugar, butter, wheat, rice, biscuits. In the absence of pancreatic juice, fermentation of carbohydrates may eventually lead to deposits of calcium oxalate; therefore the administration of **pancreatic extracts** is indicated.

PROGNOSIS.—This depends upon the intervention of complications such as diabetes, infection, hemorrhage, etc.

PATHOLOGY.—The calculus consists of an organic nucleus or matrix, composed of denuded, infected, dead, epithelial cell-remnants, mucus, leukocytes, and fatty acid crystals. Chemically, the nucleus is composed of proteins, fats, cholesterin, leucin and tyrosin. Deposited in and about this matrix are the calcium salts of carbonic, phosphoric, oxalic, and stearic acids. The calculi are seldom facetted, since they are usually not multiple and are not subject to excessive pressure. Their color is, as a rule, white and glistening. They are commonly situated near the ampulla, where the duct is the narrowest.

The ducts present the picture of a sialo-angitis. They may be fusiformly enlarged from distension (the pancreatic ranula of Virchow) or they may be ectatic or moniliform in appearance (the pancreatic acne of Klebs). The calculus may be accompanied by cyst formation, pancreatic hemorrhage, suppuration, fibrosis, atrophy, etc.

Pancreatic Cysts.—Of all pancreatic affections, cysts give the most direct physical evidences. In general, cysts are classified into two main groups: true cysts and cystoids (pseudocysts of Korte). True cysts are subdivided into retention and proliferation cysts. Cystoids are usually not directly connected with the pancreas, but arise in adjacent structures secondarily to some pancreatic disturbance. Clinically or operatively, it is often difficult to decide to which group a cystic formation in the pancreatic region belongs. Pancreatic cysts may be of congenital origin and may be associated with renal, hepatic or splenic cystic disease (dysontogenetic cysts). Parasitic (echinococcus) cysts are rare.

ETIOLOGY.—Any condition which has a tendency to obstruct the ducts, such as sialo-angitis, calculus, tumor, or fibrosis, which exerts a constriction or a traction on the ducts, is a potential factor in the formation of a cyst. Moderate obstruction of the main duct may cause a simple dilatation, the so-called pancreatic ranula of Virchow. Constriction involving the smaller ducts may cause a multiple, beaded sacculation, which Klebs has called acne pancreatica. If to the factor of mild constriction or obstruction an inflammatory, infective, or exudative element is added, a true retention cyst develops. Proliferating cysts are true neoplasms, such as cystadenoma, echinococcus, or congenital cystic disease (multiple). Pseudocysts may ensue upon a pancreatic trauma with hemorrhage followed by blood-absorption and cavity formation (hemorrhagic cyst). A pancreatic perforation, either retroperitoneal or situated near the left renal region, or in the lesser peritoneal sac, accompanied by hemorrhage, may give rise to a cyst. If the hemorrhage occurs into the lesser sac and the foramen of Winslow becomes occluded by a clot or an exudate, an omental cyst is formed.

Symptomatology.—The symptoms are identical in all types of cysts. They may be classified into three general groups: (1) pancreatic, (2) regional, and (3) general. *Those referable to the pancreas* are quite insignificant and are overshadowed and obscured by the regional symptoms. They may be those of a chronic interstitial pancreatitis. To these may be added the signs of hemorrhage, fat necrosis, and acute inflammation.

Regional symptoms depend upon the amount of pressure exerted by the cyst upon other organs or tissues. If the duodenum or the pylorus is compressed, the symptoms of gastrectasis are produced (copious retention vomiting). The portal vein may be compressed, giving the picture of hepatic cirrhosis with ascites, or, if the common bile-duct is occluded, a persistent jaundice. The symptoms of intestinal obstruction may be present either from direct mechanical pressure of the cyst, or from a paralytic ileus from interference with the mesenteric vessels, which are intimately associated with the pancreas. Pressure on the aorta produces aneurysmal symptoms, while that on the vena cava produces marked edema of the lower extremities. Cysts of the tail and body of the pancreas, due to pressure on the left ureter, may give the picture of a hydronephrosis. In addition to these regional symptoms, there may be adhesions between the cyst and other organs.

Of the *general symptoms,* pain may be prominent or entirely absent; or it may be replaced by a feeling of tension or fullness due to encroachment upon other organs, particularly upon the stomach. Pain depends upon the stretching of the cyst capsule, and upon pressure and inflammatory adhesions to adjacent organs, vessels or nerve-plexuses (girdle sensation).

Physical Findings.—Usually *inspection* shows a prominence in the left hypochondrium, situated above the umbilicus and movable with respiration.

On *palpation* a tense, sometimes tender, mass presents itself, possessing respiratory and lateral mobility. Pulsations transmitted from the aorta may be felt. The position of the cyst is little influenced by change

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of posture on the part of the patient, and possesses no spontaneous mobility.

Percussion reveals a dull area almost completely surrounded by tympany—gastric above, colonic below. It is important to remember that there is always a tympanitic area separating the mass from the liver dullness. Gastric or colonic inflation diminishes the size of the dull area as the tympany of these organs encroaches upon the cyst. Occasionally a thrill or fremitus may be felt in the absence of echinococcus cysts, when the contents of the cyst are under great tension.

Auscultation over the cyst sometimes reveals the presence of bruits and murmurs produced by pressure on the large blood-vessels (aorta and cava). These murmurs usually disappear in the knee-chest position. The size of the cyst may vary from time to time. A sudden enlargement accompanied by the signs of anemia or shock, with severe pain, may be due to a large intracystic hemorrhage caused by erosion or stretching of the vessels in the cyst-wall. A sudden disappearance or diminution in size may be due to an evacuation of the contents of the cyst into the duodenum either by way of the ducts or through perforation. This may be accompanied by a profuse blood-tinged diarrheal discharge. Disappearance of the cyst, with the signs of collapse and peritoneal irritation, may be caused by rupture into the peritoneal cavity (dullness on percussion in the flanks). The rupture of an echinococcus cyst may be accompanied by an urticaria and by eosinophilia.

X-ray studies are sometimes helpful in the diagnosis of a cyst. The duodenal loop is enlarged and compressed.

DIAGNOSIS.—Large cysts may present no subjective signs. Sometimes the cyst is a development of some other pancreatic disease (calculus, fibrosis). When a cyst is of sufficient size to become accessible to palpation, four general diagnostic possibilities present themselves:

1. Is the mass in the abdominal wall?
2. Is it intraperitoneal?
3. Is the mass retroperitoneal?
4. Is it pancreatic?

(1) *Conditions of the abdominal wall* which must be considered are, marked diastasis of the upper recti with protrusion of the stomach and colon, especially when the patient attempts to assume the sitting from the reclining posture, large epigastric hernias, encapsulated abscesses, and certain tumors, such as lipomas, rhabdomyomas, Nelaton's tumors.

(2) *Intraperitoneal cystic enlargements* would include cysts of the liver and spleen, gall-bladder enlargement, encapsulated peritoneal exudates (ascites), tuberculosis or malignancy, and gastric, intestinal, mesenteric, omental, and ovarian cysts.

(3) The *retroperitoneal enlargements* which may give rise to confusion are the renal cystic tumors, hydronephrosis, and suprarenal cysts and tumors (hypernephroma with cystic degeneration). Aneurysms, cold abscesses (spinal disease), retroperitoneal sarcoma (Lobstein's cancer), and abdominal Hodgkin's disease are possibilities.

(4) The evidence in favor of the *pancreas* as the site of origin consists in the position of the cysts. By far the largest number of pancreatic cysts present themselves between the greater curvature of the stomach and the transverse colon. A mass in this region surrounded by an area of tympany, possessing slight respiratory, little lateral, and no postural or spontaneous mobility, directs the attention to the pancreas. Variations in the size of the mass, which may also occur in a coëxisting hypdronephrosis, and pulsations and bruits which disappear in the knee-chest posture, are important evidence. The pancreas is in such intimate relationship with the aorta that the pulsations of the latter are transmitted to the cyst. As the pancreas falls away from the aorta in the prone posture, these pulsations disappear. This is an important differential point in the exclusion of aortic aneurysm, in which the pulsations and bruits persist irrespective of the posture.

Aspiration of the cyst with an examination of the contents for pancreatic ferments is too risky to be utilized for diagnostic purposes. The method is furthermore of doubtful utility.

A large, distended gall-bladder is sometimes difficult to differentiate from the gastrohepatic type of pancreatic cyst. The latter is separated from the liver by an area of tympany, while the dullness of gall-bladder enlargement merges with that of the liver. In rare cases the hepatic flexure of the colon is hooked about the neck of the gall-bladder, and then the latter is also separated from the liver dullness by a tympanitic band. In such a type of case one can resort to the method of Stengel's auscultatory percussion for the determination of the origin of the mass. Large gall-bladders move downward, forward, and inward with respiration, and do not permit of expiratory fixation. Cysts of the left lobe of the liver, abscesses, and degenerated gumma must be considered. The high position of the left leaf of the diaphragm can not be utilized in differentiation, as it is also elevated by pancreatic cysts. Omental cysts must be taken into consideration. Cysts of the mesentery may occupy the site in which pancreatic cysts usually occur. They are situated about the umbilicus and usually present what is known as Tillaux's triad. This consists of (1) excessive mobility, (2) dullness, and (3) a surrounding tympanitic area with a small tympanitic band crossing the dull area. Omental cysts are usually accompanied by peritoneal irritation and ascites. Exudates into the bursa omentalis are the result of gastric or pancreatic disease and give the signs of cysts (cystoids). In diagnosing the low or infracolic type of cyst, pregnancy, ovarian cysts, urachal cysts, and distended urinary bladder must be considered. Pelvic examination would determine the origin of the cyst. Encapsulated peritoneal exudates are usually secondary to perforation of the stomach, gall-bladder, or appendix, or to a tuberculous peritonitis. There is usually a history of the antecedent disease. The mass is tender, immobile from inflammatory adhesions, and characterized by rigidity of the overlying musculature. Splenic cysts can be differentiated by the fact that the spleen is never covered by the colon. They are pushed upward by colonic inflation and downward when the stomach is distended, but are never covered by a tympanitic area, except in perforative peritonitis. There may be evidence in the blood of splenic disease. Cysts and tumors of the intestinal tract are very movable, and change their position spontaneously (peristalsis). Radiographic examination may be of some help in localization.

A hydronephrosis on the left side is difficult to differentiate from a cyst of the body or tail of the pancreas. Both conditions may exist simultaneously, due to the pressure of a pancreatic cyst on the left ureter. A hydronephrosis resembles cysts in its tendency to disappear rapidly. This disappearance may be accompanied by polyuria. A simple hydronephrosis is retroperitoneal, and becomes tympanitic on inflation of the colon. Cold abscesses from renal or spinal disease must be excluded (dorsal or lumbar Pott's disease).

When suppuration begins in a previously unsuspected cyst it is impossible to make a correct diagnosis.

COMPLICATIONS.—The three prominent complications are hemorrhage, perforation, and thrombosis. Diabetes due to pancreatic fibrosis may occur. Occasionally the cyst suppurates or undergoes malignant degeneration. Acute hemorrhagic pancreatitis and fat necrosis are complications which may first direct the attention to the presence of an unsuspected cyst. In very rare cases, when cysts are situated in the tail of the pancreas, they may be pedunculated and freely movable, and may undergo torsion.

TREATMENT.—The treatment is entirely **surgical** as soon as the diagnosis is established. It is instituted with the view of sparing the patient the possibility of a subsequent complication if the cyst is permitted to grow. Cysts of the tail of the pancreas are amenable to complete **extirpation** if not bound down to adjacent structures by adhesions. Recurrences are possible if the etiological factor is an irremediable one (pancreatic fibrosis).

PROGNOSIS.—The prognosis depends upon the pressure signs, the amount of pancreatic involvement, and the complications. Interference with alimentation either by involvement of the gastro-intestinal or biliary tract or by the occurrence of diabetes seriously affects the prognosis.

PATHOLOGY.—In the gross the cysts vary in size, the largest encroaching upon the greater part of the abdomen. The true cysts are usually smooth and are not as a rule adherent to adjacent organs. Cystoids usually have a thick, inflammatory capsule and are adherent to neighboring organs and structures. True cysts usually have some duct or alveolar epithelium lining and may communicate with one or more ducts. Pseudocysts have no epithelial lining. The epithelium may disappear either by pressure atrophy from tension, or by the digestive action of the contents of the cyst from disturbance of the circulation in the cyst-wall, or from an inflammatory reaction. The cyst may contain inactive pancreatic juice secreted by the pancreatic cells lining the interior of the cyst, or originating in the communicating pancreatic tissue; or they

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may be hemorrhagic or purulent if inflammation and hemorrhage occur. As the fluid accumulates, the tension in the cyst increases, and it grows in the direction of least resistance. Reference to the regional anatomy will explain the direction of cyst growth. The usual site is between the stomach and the transverse colon, the gastrocolic omentum forming part of the capsule anteriorly. This type of cyst is known as the gastrocolic or supracolic cyst. Occasionally cysts grow between the layers of the transverse mesocolon (mesocolic cysts), sometimes below the colon (subcolic cysts), and, in rare cases, between the lesser curvature of the stomach and the liver. The last type occurs in cases of gastroptosis or hepatic atrophy. The gastrohepatic omentum then forms the anterior layer of the cyst-wall. Occasionally cysts appear suddenly or begin to grow rapidly, particularly the cystoids. This is usually due to an extensive hemorrhage into the cyst produced by the digestive action of the contents on the blood-vessels. At times large cysts suddenly disappear, and the contents are evacuated into the duodenum through the ducts, or, by perforation, into adjacent organs or structures. Cysts may undergo malignant degeneration, calcification, rupture, or replacement fibrosis. The contents are usually composed of blood-proteins, hemoglobin, peptones, mucin, cholesterin, fatty acid crystals, cell-detritus, leukocytes, and inorganic salts.

The contents of pancreatic echinococcus cysts are usually albumin-free and contain succinic acid, large amounts of sodium chlorid, sugar, and hooklets. In addition to the formed elements, true cysts may contain one or more of the pancreatic ferments. Occasionally the ferments disappear in old cysts. Their presence is not, however, pathognomonic of the pancreatic origin of a cyst, since both proteolytic and lipolytic ferments have been demonstrated in other cystic fluids. Pus-cells secrete a tryptic ferment. The presence of a starch-splitting ferment has some significance, although it can rarely be demonstrated.

The pathology of cystoids is usually a hemorrhagic disruption of pancreatic or adjacent tissues with digestion of the clot, either by the liberated pancreatic juice or by autolysis, with the formation of a cavity. There is an inflammatory reaction about this area, accompanied by a reactive encapsulating fibrosis circumscribing the lesion and producing a thick, connective-tissue capsule, which has no epithelial lining.

Diseases of the Pancreatic Blood-Vessels

Hemorrhage into the pancreas proper is so intimately associated with acute hemorrhagic pancreatitis, that clinically it is almost impossible to differentiate between the two conditions. A pancreatic hemorrhage may be followed by an inflammatory reaction (autolytic or infectious), or an inflammatory lesion may be associated with a hemorrhage, making it quite impossible to determine cause and effect.

Etiology.—Men who are obese and alcoholic are predisposed to these diseases. The usual causes are trauma, diseased blood-vessels, diseases of the blood, and infections. A hemorrhage following trauma may be due to a penetrating wound (stab) or to compression of the pancreas against the unyielding spinal column (contusion or laceration). Occasionally preagonal or postmortem autodigestion is accompanied by bleeding. Arterial disease, aneurysm, arteriosclerosis, and stasis (cardiac, portal, and pulmonary), severe infections, as hemorrhagic smallpox, measles, and septic endocarditis, are the usual causes. The condition may also follow operations in the region of the biliary passages. The various blood diseases, as pernicious anemia, leukemia, as well as scurvy, and poisoning with phosphorus and mercury, are occasional etiological factors. Conversely, there is a hemorrhagic tendency in pancreatic diseases, particularly in carcinoma with jaundice and interstitial pancreatitis. Experimentally, the injection of the duodenal contents (enterokinase) into the ducts converts protrypsin into trypsin, which digests all of the pancreatic tissues except the connective tissue. This is accompanied by hemorrhage.

Symptomatology.—The symptoms are so closely associated with those of acute hemorrhagic pancreatitis that, for convenience, they will be discussed under that heading. The same applies to the diagnosis and treatment.

The various pathological changes which occur in the blood-vessels of the islands of Langerhans (hyalin degeneration, etc.) are of some significance in the production of diabetes.

Pathology.—The pancreas is enlarged, firm, and of a purple hue. The peritoneum may be blood-stained, there may be a perirenal extravasation of blood on the left side, or there may be bleeding into the lesser peritoneal sac. If the hemorrhage is small it may be entirely absorbed, and the only evidence at operation or postmortem examination may be the presence of a pigmentary deposit (hemosiderin). The hemorrhage is sometimes followed by a replacement fibrosis, by cyst formation, or by suppuration with the formation of an abscess. The hemorrhage may initiate a chronic interstitial pancreatitis.

DISEASES OF THE PANCREATIC PARENCHYMA

For convenience of clinical description these conditions may be classified as:

1. Inflammatory—acute, hemorrhagic pancreatitis.
2. Degenerative—fat necrosis.
3. Neoplastic.

Acute Hemorrhagic Pancreatitis.—Of all the acute abdominal diseases this is one of the most fulminating and serious. Dieulafoy has aptly termed it "the acute pancreatic drama." It requires early recognition and prompt operative interference in order to prevent a fatal termination. It is only since the development and increased frequency of operations in the gall-bladder region, that our attention has been focussed upon this disease. Previous to its recognition the deaths

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resulting from this disease were reported as "acute indigestion," or "acute cardiac failure."

Etiology.—The predisposing causes are the triad: obesity, alcoholism, and a history of gall-stones. The general causes may be classified as:

1. Trauma—*see* pancreatic hemorrhage.
2. Obstruction to the flow of the pancreatic juice such as that caused by swelling of the papilla in duodenitis, sialo-angitis, pancreatic and biliary calculi, and by other conditions which obstruct the ducts, such as scars, cysts, and neoplasms.
3. Infectious diseases, as mumps, typhoid and influenza.
4. Vascular degeneration (syphilis) and emboli.
5. Extension from inflammatory processes in adjacent organs, as in the case of perforated gastric and duodenal ulcers.

The most frequent cause is the lodgement of a calculus (biliary) at the papilla, thereby converting the two separate channels of the bile and pancreatic juices into a common opening, and permitting easy access of the bile into the pancreatic tissues. This has been demonstrated by Opie in a series of cases at the Johns Hopkins Hospital. The disease has also been experimentally produced by the injection of various substances into the pancreatic ducts, such as bile-salts, duodenal contents, hydrochloric acid, oil, and bacteria of the colon-typhoid group, etc. Experimentally, the production of a pancreatic trauma has in some cases been followed by a pancreatitis. The entrance of bile and duodenal contents into the ducts activates the pancreatic juice, and this in turn produces autolysis. If to this the element of infection is added, the requisites for an acute hemorrhagic pancreatitis are complete. Occasional cases have been reported in which, in excessive vomiting, there was a regurgitation of duodenal contents into the ducts due to reverse peristalsis and aspiration.

The arrangement of the ducts has some influence upon the incidence of pancreatitis. Under Anomalies of the Ducts the patency of Santorini's duct was considered. If this duct is unoccluded and an ascending infection of Wirsung's duct occurs, the pancreas may still empty its contents into the duodenum, and a stasis may thereby be averted. If the lesser duct is also impervious, there is a greater tendency toward pancreatic lesions in gall-stone disease. If the common bile and pancreatic ducts open separately into the duodenum, there is less likelihood of pancreatic disturbance in cases of common duct-stone.

The four main paths by which the pancreas may be involved may be summarized as follows:

1. Enterogenous (duodenitis).
2. Biliary.
3. Hematogenous (mumps and typhoid).
4. By direct extension from inflammations in contiguous organs.

A lymphatic origin has not been definitely demonstrated, although Deaver considers this the most likely path of infection.

SYMPTOMATOLOGY.—It is impossible to clinically differentiate the three stages. Usually one is able to elicit a previous history of gastrointestinal or gall-bladder disease. Some patients volunteer the information that they are suffering from another attack of "acute indigestion," which seems to be a little different and more severe than the previous attacks. The disease may follow a recent operation on the biliary passages, such as a cholecystectomy or cholecystotomy. It is also advisable to inquire of the patient whether there has ever been an attack of jaundice (cholangitis). In some cases, there is no history of any previous disease. The possibility of a previous acute attack of mumps with severe abdominal pains and vomiting might indicate that the pancreas was implicated. This may cause increased susceptibility to subsequent infections. In some cases there is no demonstrable previous disease, and then it is well to look for the evidences of furunculosis or of tonsillar infection. The most characteristic feature of the attack is the suddenness of the onset. The attack is usually attributed by the patient to some article of food ingested at the previous meal. He is seized with a very severe, agonizing epigastric or umbilical pain, scarcely yielding to good-sized doses of morphin. The pain is usually sharp, piercing, stabbing, or colicky, and radiates toward the back, particularly to the left. In some cases the pain is primarily intense in the back and radiates forward. Sometimes the paroxysm resembles a gall-stone attack. The severity of the pain can only be explained by the involvement of the semilunar ganglion in the hemorrhagic exudate, or by the excessive tension created in the pancreas by the bleeding.

Closely following the onset of the pain is the marked collapse and shock. It can hardly be attributed to the amount of blood lost, as this may be practically negligible. It depends upon three factors: splanchnic paralysis due to involvement of the large ganglia (exemplified in the Goltz shock experiment in the frog), implication of the cardiac sympathetic nerve, and finally upon the absorption of trypsin into the general circulation. Experimentally, it has been produced by the latter method. Intractable vomiting soon makes its appearance, accompanied by bleeding from the stomach due to the rupture of vessels from the retching, or from a general hemolytic condition, which may develop in pancreatic disease. Occasionally a pancreatic hemorrhage empties itself into the duodenum and is expelled by a hematemesis. Constipation is the rule, due to the development of paralysis of the ileus. Salivation is rare. Singultus may be a prominent symptom.

Physical Findings.—Inspection shows a marked cyanosis of the lips, as contrasted with the extreme pallor of the face. The pupils are usually dilated, the expression drawn and anxious. The body is covered with a cold clammy perspiration. The respirations are superficial and shallow, due to the high position of the diaphragm (abdominal distention) and to a possible acidosis. The pulse is exceedingly rapid, of poor quality, and thready. The temperature is usually of a septic type. The abdomen is markedly distended, with upper abdominal rigidity or a doughy sensation on palpation. The distention is intractable to enemas and internal therapy. There is a diffuse, extreme abdominal tenderness.

No peristalsis is visible, and little gurgling is discernible on auscultation with the stethoscope (paralytic ileus).

If the hemorrhage is extensive, dullness may be elicited on percussion of the flanks (hemoperitoneum).

Some observers report their ability to palpate a swollen, tender, pulsating pancreas. This seems hardly conceivable when one reflects upon the abdominal distention, tenderness, and rigidity, which are usually present. It may be possible to hear an aortic bruit. The desperate condition of the patient, however, seldom permits extensive palpatory or postural manipulations.

In addition one may note signs of acute gastric dilatation, due either to obstruction of the pylorus by a swollen pancreatic head, to toxemia, or to peritonitis which rapidly supervenes. If it is possible to overcome the constipation, the resulting bowel movement may be blood-tinged, due to bleeding into the duodenum, to perforation, to hemolysis, or to thrombosis of the mesenteric vessels. Since the splenic vein is in close proximity to the pancreas this may become thumbased with a resultant splenic enlargement which may temporarily mask the primary pancreatic condition.

The blood shows a high leukocytosis, and increased bleeding and coagulation time. The urine is usually scanty or suppressed, and it may be possible to demonstrate a lipase. Acetone and diacetic acid may be present. The presence of a large number of calcium oxalate crystals may be of some significance, since in pancreatic disease there is sometimes an increased calcium output. This may explain the tendency toward bleeding which exists in this disease. The Cammidge pancreatic reaction, upon which so much hope was placed at one time, has been permanently abandoned as unreliable. It depended upon the demonstration of a pentosazon. Occasionally glucose has been demonstrated in the urine.

Diagnosis.—A previous history of icterus, gall-stones, gall-bladder operation, and alcoholism may be of some diagnostic aid, particularly in the case of an obese patient. A recent infection, typhoid, mumps, or influenza, strengthens the suspicion. The presence of a glycosuria, and a gall-stone history, certainly indicates a disturbance of the pancreas.

From a differential standpoint it becomes necessary to consider the following possibilities:

1. *An incarcerated epigastric hernia.*
2. *Acute gastric dilatation.* This condition appears early in pancreatitis, but occurs also postoperatively, and following severe infections, in peritonitis and intestinal obstruction.
3. *Ptomain or mineral poisoning* (bichlorid of mercury).
4. *Gastric or duodenal perforation.* Here a previous history of gastric or duodenal ulcer may be obtainable. The signs of a pneumoperitoneum may be present. It must not be overlooked that a posterior

perforation of the stomach or duodenum may initiate a pancreatic inflammation by extension.

5. *Cholelithiasis, gangrene, and perforation of the gall-bladder.* The presence of bile-pigment in the urine, tenderness over the gall-bladder point, tenderness to the right of the tenth dorsal vertebra (Boas gall-bladder point), and tenderness of the outer third of the trapezius muscle, may all be present. The pain usually yields to narcotics. This gall-stone colic is sometimes the precursor of a subsequent pancreatitis, which may follow at once or at a later period.

6. *Pancreatic calculus.* This is not clinically distinguishable from pancreatitis, but even if it were, this would be of no practical value from a surgical standpoint, as both conditions demand prompt surgical interference.

7. *Attacks of angina pectoris,* particularly those accompanied by acute gastric dilatation and collapse. The short duration of the attack and the response to nitrites is important in differential diagnosis.

8. *Spasm of the mesenteric arteries* (dyspraxia arteriosclerotica abdominalis of Ortner). This condition may give all the signs of a pancreatitis. Here the demonstration of an arteriosclerosis, and of a hyperpiesis, and the relief of the attack by nitrites and morphin should clear up the difficulty.

9. *Mesenteric thrombosis*—severe intestinal colic accompanied by bloody evacuations with tenesmus, and the history of thrombosed hemorrhoids, intestinal ulceration, etc. This condition may also be a sequel to pancreatitis.

10. *Thrombosis of the splenic vein and infarcts of the spleen.* The presence of splenic enlargement and the history of an infection (typhoid) or leukemia should be looked for. This condition should also arouse the suspicion of a disturbance of the pancreas as the etiological factor.

11. *Aneurysm of the abdominal aorta, which has ruptured.* The symptoms are dullness in the flanks, expansile pulsation, and rapid exitus with the picture of an internal hemorrhage.

12. *Intestinal obstruction.* Here the diagnosis may become exceedingly difficult, particularly when the obstruction is high, as in the case of a hernia in Treitz's fossa, in the foramen of Winslow, or in the diaphragm. This type of obstruction is usually mechanical and differs from the paralytic type, which is more characteristic of pancreatic inflammation. An annular inflamed pancreas may, however, obstruct the duodenum by its pressure.

13. *Perirenal abscess on the left side*—positive Murphy or Brewer sign with edema of the left flank. The same applies to a pyelitis (pyuria).

14. *An acute hematogenous infection of the left kidney.* This is usually secondary to a furunculosis or to tonsillar infection.

15. *Hemorrhage into the left adrenal.* This can scarcely be differentiated from pancreatitis.

16. *A perforation of the undescended retrocolic appendix.*

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17. *A ruptured ectopic with free blood in the peritoneum, with intestinal irritative symptoms.* This may for a time be a diagnostic consideration.

18. *Neuralgia of the celiac plexus from aneurysm or spondylitis* (Pott's). This condition, and the crises of locomotor ataxia, angioneurotic edema, exophthalmic goiter, and lead-poisoning may temporarily cause confusion.

COMPLICATIONS.—The complications may be peritonitis, perforation into the stomach or bowel, thromboses of the veins of the portal system, or a perirenal hemorrhage on the left side.

TREATMENT.—Treatment is entirely surgical. When the diagnosis is in doubt, early exploratory **laparotomy** is justifiable. The symptoms are so threatening and severe that no time should be sacrificed in awaiting the development of definite signs. As palliative measures, large doses of **morphin** and **atropin**, the combating of shock, and perhaps a preliminary **blood-transfusion** may be indicated. Spontaneous recovery by perforation and drainage into the bowels has been reported.

PROGNOSIS.—The outlook is always extremely grave. Death usually occurs within a week, unless operative interference is undertaken. The patient may not, however, be able to withstand the shock at the onset, and may succumb within a period of twenty-four hours. After the first week an abscess of the pancreas may form, which sometimes ruptures into other organs. There are on record unmistakable cases of pancreatitis revealed at subsequent operation or at autopsy by a pigmentary deposit with fibrosis. The case may terminate in peritonitis, in thrombosis of the splenic vein, or in sepsis.

PATHOLOGY.—Three types of inflammation have been described: the hemorrhagic, suppurative, and gangrenous. These are not in reality three distinct pathological entities, but are only progressive changes of the same disease. No hard and fast line can be drawn between the various stages. The transition from one into the other is only a matter of time. If the patient survives the acute inflammation, the condition may later become suppurative or gangrenous. Macroscopically, the pancreas is usually enlarged and has a black hue, and some turbid or blood-stained peritoneal fluid is present. Most characteristic of all is the presence of small areas of fat necrosis which stud the omentum, mesocolon, and mesentery. These will be discussed in detail under the section on Fat Necrosis. Occasionally some doubt exists as to the differentiation between pancreatitis and preagonal or postmortem autolysis. In the latter condition the pancreas is pale and white, has a ground-glass appearance, and stains diffusely with eosin (Chiari).

Microscopically, little of the cellular substance of the involved pancreatic area remains except some cell-detritus. The nuclei are digested, and the interstitial tissues are infiltrated with blood from congestion and the erosive action of the ferments on the blood-vessels. As the disease progresses, the pancreas may suppurate (pancreatitis apostemotosa) or become gangrenous. A primary suppurative pancreatitis may follow portal sepsis or septic endocarditis. There may be thrombosis of the splenic or mesenteric veins, due to their close proximity to the pancreas.

Pancreatic Abscess.—This condition usually follows the acute hemorrhagic condition. The *etiology* is the same. The rupture of a gastric ulcer may produce a peripancreatic or pancreatic abscess. Occasionally pancreatic cysts suppurate and produce abscesses. It has a prolonged course and shows the picture of a sepsis. A palpable, doughy mass may present itself in the epigastric region. Fluctuation may be present. *Treatment* is purely surgical.

Fat Necrosis.—The presence of this condition is the only pathognomonic evidence of pancreatic pathology.

ETIOLOGY.—The fact that this lesion is usually limited to the fatty tissues about the neighborhood of the pancreas led many early observers to assume that fat necrosis had some relationship to the action of pancreatic lipase on these tissues. Experimentally this has proved to be the case. After ligation of both pancreatic ducts, with simultaneous occlusion of the pancreatic veins to prevent absorption of the pancreatic juice, the typical lesion of fat necrosis has been produced. A direct incision into the pancreas, which permits the escape of the pancreatic juice, produces the lesion wherever it comes into contact with the tissues. Obstruction of the pancreatic ducts favors a damming of the juice into and between the alveoli and into the lymphatics and adjacent tissues. Areas of fat necrosis have been found at some distance from the pancreas, as in the pericardial fat. To explain such a lesion one must invoke the theory of metastasis of pancreatic cells or secretion, by way of either the blood or the lymph stream. This may also be a satisfactory explanation for the presence of the necrotic hepatic areas which are sometimes found. This is the pancreatic embolic or metastatic theory of Payr and Martine.

The etiology of fat necrosis coincides with that of acute hemorrhagic pancreatitis. Fat necrosis is a manifestation of that condition. The lipolytic pancreatic pro-enzyme is activated by the bile, which gains access to the ducts. The obese are predisposed because of the excessive peripancreatic fat infiltration. Just as jaundice is frequently an evidence of biliary obstruction, fat necrosis is unmistakable evidence of pancreatic obstruction or stasis. Pilocarpin injections, in the presence of stasis, favor the development of necrosis by an increase of the tension in the ducts.

SYMPTOMATOLOGY.—The symptoms are usually those of an acute hemorrhagic pancreatitis. Necrosis is only an element or a result of pancreatic inflammation. If in the absence of any other definite signs the candle-grease spots of necrosis are found at laparotomy, the diagnosis of pancreatic disease becomes at once established. In rare cases, the presence of a lipolytic ferment in the urine as demonstrated by Opie according to the method of Hewlitt, may be of some diagnostic value. This method depends upon the principle that fat-splitting ferments decompose a neutral solution of ethyl butyrate into butyric acid, which imparts an acid reaction to the medium.

Diagnosis.—At operation, the spots of fat necrosis may be mistaken for tubercles or carcinoma nodules. The absence of elevation is evidence against the presence of either of these two. The differential diagnosis, prognosis, and treatment are those of acute hemorrhagic pancreatitis.

Pathology.—On opening the abdomen either operatively or at postmortem, the attention is immediately directed to the presence of small, opaque, candle-grease spots studding the fat of the abdominal walls, omentum, mesentery, and peritoneum. They are usually about the size of a split pea. To the inexperienced the lesions may resemble miliary tubercles which have undergone caseous degeneration, or small carcinoma nodules. These nodules are surrounded by a hyperemic or hemorrhagic zone, either of inflammatory or of vascular origin. In cases associated with jaundice they may have a yellow tinge. Microscopically, the lesion consists of a central area devoid of structure and composed of cell-detritus produced by the action of the lipase on the fatty tissue. The latter is decomposed into fatty acid and glycerin, the acid combining with the calcium, which is present in the blood, to produce a soap of calcium stearate or oleate (candle-grease appearance). The glycerin is absorbed. Microchemically, the addition of sulphuric acid forms long calcium sulphate crystals. Benda's stain, which is a half-saturated solution of copper acetate, imparts a green color to soaps. Normal fatty tissues (neutral fat) are unaffected. Throughout the areas of fat necrosis, strands of connective tissue unaffected by the action of the pancreatic juice may be seen. In addition, needle-shaped crystals, of fatty acid, red blood-cells, and leukocytes may be demonstrated. Toward the periphery of the lesion an inflammatory reaction is found, with a proliferation of the fixed tissue-cells.

Tumors of the Pancreas.—With the frequency of operations on the biliary passages, the opportunities for palpating the head of the pancreas have increased, and the number of diagnoses of pancreatic malignancy have correspondingly decreased with the recognition of the fact that the head of the pancreas often becomes indurated in infective conditions of the biliary passages.

Symptomatology.—An early sign of carcinoma of the head of the pancreas is a severe, progressive, intense jaundice, usually the so-called black or melanotic jaundice, produced by obstruction of the common duct. So complete is the obstruction that no stercobilin can be demonstrated in an examination of the feces. The obstruction remains permanent, a circumstance which hardly ever occurs in the obstructions produced by calculi. Pain is usually a constant symptom and is caused either by obstruction of the ducts, inflammation, or by pressure on the celiac ganglia. There is marked anorexia, with a profound distaste for fats and meat. Vomiting may be a constant feature. The patients complain of a rapid and excessive loss of weight and of extreme weakness.

On physical examination the most striking feature after the jaundice

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is the marked cachexia. The severest type occurs in this disease. There are several factors which enter into its production: the anorexia, vomiting (obstruction), diarrhea from lack of fat digestion, and absorption (obstruction of the bile-duct), and diabetes, which may occur as a complication. The temperature is usually subnormal. The abdomen may be distended by an ascites. The presence of the latter, with signs of complete obstructive jaundice, indicates pancreatic neoplasm. The ascites may also be due to a metastatic cancerous peritonitis. In severe emaciation and in cases of ptosis the head of the pancreas may be felt as a hard, nodular, immovable mass. With gastric inflation the tumor usually disappears. The tumor may transmit aortic pulsations. If it is pyramidal in shape, the pulsations may resemble the expansile pulsations of aortic aneurysm. If the receptaculum chyli is involved, there are evidences of chylous ascites and enlargement of the supraclavicular gland on the left side (Virchow's gland). An enlarged, painless gall-bladder may be felt. The gastric contents may show the presence of an achylia gastrica. An examination of the feces may reveal the presence of blood, a creatorrhea, and a steatorrhea (neutral fat and absence of soaps and fatty acid crystals). The urine usually shows the absence of indican and the presence of a large amount of calcium oxalate crystals, and occasionally a glycosuria. The Loewi adrenalin pupillary test may be positive. The Bard and Pick syndrome consists of a painless gall-bladder enlargement, with a progressive, permanent jaundice, and inanition and cachexia. These observers attribute the syndrome to an invasion of the ampulla by a pancreatic or choledochus neoplasm.

Diagnosis.—The principal conditions to be differentiated are: the impaction of a calculus in the common duct, and carcinoma of the ampulla, duodenum or ductus choledochus. Evidence of a calculus would be the rather sudden onset of the jaundice following an attack of colic. The jaundice is seldom absolute, and is usually accompanied by a febrile paroxysm (Charcot's biliary fever). The gall-bladder is usually tender and contracted, and shows evidence of inflammation (adhesions). In pancreatic neoplasm it is enlarged and not tender (Courvoisier's law).

Pyloric and intestinal tumors also come into diagnostic consideration. Pyloric neoplasms usually possess respiratory, postural, and spontaneous mobility, while pancreatic tumors are fixed. Intestinal tumors are very movable. Radiographic examination is of some aid in the differential diagnosis. Clinically it is impossible to differentiate ductus choledochus tumors. Retroperitoneal sarcomata and Hodgkin's disease may, in the beginning, give rise to difficulty. Tumors of the tail of the pancreas may resemble splenic, renal, adrenal, and gastric tumors. Horse-shoe or fused kidneys may be mistaken for a pancreatic mass. For differential diagnosis of other masses, see Pancreatic Cysts.

Benign, tuberculous, specific, and inflammatory tumors of the pancreas may be difficult of diagnosis. Occasionally Hodgkin's disease or leukemia produces infiltrative pancreatic lesions. If the diagnosis is once definitely established, death usually occurs within a period of from six months to a year. There are cases on record where complete extirpation of the tumor has resulted in recovery.

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COMPLICATIONS.—The condition may be complicated by hemorrhage, perforation, and metastases.

TREATMENT.—Treatment may be divided into palliative, symptomatic, and surgical. It may be necessary to resort to large doses of **morphin** to keep the patient comfortable. The pruritus is often a distressing symptom; it may be controlled by local applications (**menthol, carbolic**) or by **antipyrin, sulphonal**, etc. For the control of diarrhea, a **restriction of the fats**, with the exception of milk fat, is indicated. The various pancreatic preparations which may be used will be fully dealt with in the next section. For the persistent jaundice, **cholecystenterostomy** may be resorted to, and for the signs of pyloric obstruction it may be necessary to perform a **gastro-enterostomy**. These cases are usually poor surgical risks on account of the hemorrhage and shock. The former may be controlled by prophylactic doses of **calcium chlorid** or **lactate**, as recommended by Cammidge and Robson, or by a **transfusion**. Tumors of the tail of the pancreas lend themselves to complete **extirpation** when not complicated by adhesions.

PATHOLOGY.—Tumors of the pancreas are not of frequent occurrence. The chief type of tumor is the carcinomata. It may be primary, metastatic, or due to extension from adjacent carcinomata of the stomach, duodenum, or biliary ducts. The usual type is the scirrhus, then the encephaloid, with occasionally a colloid degeneration. Many cases diagnosed at one operation as scirrhus, have been found at subsequent operations to have disappeared under biliary drainage. These were clearly cases of induration of the head of the pancreas from interstitial pancreatitis secondary to a biliary stasis. The tumors may show evidence of hemorrhage, degeneration, fibrosis, or biliary pigmentation.

The usual site of the tumor is in the head of the pancreas. In may originate in the epithelium of the ducts or alveoli. The adenomatous type of tumor may be accompanied by cyst formation—the cyst-adenoma. Sarcomata are rare among tumors of the pancreas. From the topography of the pancreatic head, tumors in this region usually involve adjacent structures. The duct of Wirsung is compressed, with a resulting pancreatic fibrosis, cyst formation, or hemorrhage. In a large percentage of cases the common bile-duct runs through the head of the pancreas and can hardly escape compression, with the resulting symptoms of complete biliary obstruction. Pressure on the duodenum may cause gastrectasis and the signs of intestinal obstruction. Portal, mesenteric, and caval involvement are frequent. In addition, the tumor forms adhesions or perforates into the stomach, duodenum, or colon.

DISEASES OF THE STROMA

Chronic Interstitial Pancreatitis

Etiology.—There are several main causes for proliferation of the interstitial tissues of the pancreas:

(1) Vascular, as from arteriosclerosis, hemorrhage, stasis (pulmonary, cardiac, portal), and hemochromatosis.

(2) Primary pancreatic disease, as a sequela to an inflammation, cyst, calculus or carcinoma, or any obstructive lesion occluding the ducts.

(3) Hepatic, as in portal and biliary cirrhosis and cholelithiasis.

(4) Toxic and infectious, as in alcoholism, typhoid, and syphilis.

(5) By extension from perigastric adhesions (linitis plastica).

The disease has been experimentally produced by the injection of various irritants into the ducts, such as tuberculin, bacteria, etc.

There are two chief types of pancreatic cirrhosis. Opie has classified them as:

A. Chronic interlobular pancreatitis.

B. Chronic interacinar pancreatitis. This type is of special significance in the pathology of diabetes.

Chronic Interlobular Pancreatitis.—PATHOLOGY.—The organ is firm and compact, the markings of the lobules are accentuated, and the main ducts are usually dilated. This type commonly follows an obstruction of the ducts. Between the lobules there is an overgrowth of connective-tissue, which exerts traction or congestion on the smaller ducts, thereby predisposing to cyst formation. The cells of the alveoli become atrophic, due either to the increased pressure within the ducts or to the presence of fibrous tissue surrounding the ducts. There may be a replacement fibrosis with a lipomatosis. This type of pancreatitis is analogous from an etiological and pathological standpoint to a hypertrophic biliary cirrhosis (Hanot's). Since the interacinar tissue is not connected with the ducts except developmentally, the islands of Langerhans are usually spared in this type of pancreatitis. The common bile-duct, which is so intimately connected with the head of the pancreas, usually undergoes compression.

SYMPTOMATOLOGY.—Both types will be discussed together. The symptoms may be entirely masked by the primary hepatic or biliary disease. The onset is slow and insidious, and begins with anorexia and loss of weight. There is usually a disgust for meats and fats. There may be pancreatic vomiting and diarrhea (see General Diagnosis) with the usual findings of pancreatic deficiency. The symptoms of an achylia gastrica may be associated with the other signs. If the common duct is compressed, jaundice may be a prominent feature (see Tumors of the Pancreas, p. 53. There may be a glycosuria, or the sugar tolerance may be diminished when the islands become involved. Occasionally the liver and spleen are enlarged. There may be pigmentation of the skin and mucous membrane (bronzed diabetes).

DIAGNOSIS.—Differentially, the following conditions must be eliminated:

(1) Sprue or psilosis. Sir Patrick Manson has called attention to this tropical disease, which gives evidences of pancreatic deficiency. The presence of ulceration in the mouth (Crombie's ulcers), a tropical history, and a ready response to treatment with strawberries and raw milk and santonin, render the diagnosis certain. It may be possible to demonstrate the monilium of Ashford in the stools.

(2) Pernicious anemia, diagnosed from the blood-picture.

(3) Banti's disease, differentiated by splenic enlargement, leukopenia, epistaxis, etc.

(4) Pick's adhesive pericardial syndrome.

(5) Pellagra, characterized by nerve disturbances and the heliotropic pigmentation.

(6) Chronic malarial cachexia, differentiated by demonstration of the parasite, and by therapeutic response.

(7) Addison's disease, showing low blood-pressure, pigmentation, and elevation of pressure shortly after the injection of adrenalin (Greenbaum's test).

(8) Linitis plastica with secondary pancreatic involvement.

(9) Tabes mesenterica.

(10) Amyloid disease and its various causes (syphilis and suppuration).

(11) Severe diabetes.

(12) Carcinoma of the head of the pancreas.

(13) Neoplasms of the bile-ducts and impacted stone at the papilla.

COMPLICATIONS.—The condition is complicated by acute hemorrhagic pancreatitis, fat necrosis, and cyst formation.

TREATMENT.—Prophylactically, the treatment of gall-stone disease and syphilis may prevent the development of a pancreatic fibrosis. Dietetically, fats and animal proteins must be restricted. Deficient natural pancreatic digestion may be supplemented by the administration of **pancreatic products**. The following preparations have been used:

(1) *Holadin* (whole pancreas) with or without bile-salts in capsule form, or pancrobilin.

(2) *Pancreatic extract with calcium carbonate* in salol-coated capsules.

(3) *Pancreone,* which is a tannate of the pancreas insoluble in the stomach.

If there is a deficiency of hydrochloric acid in the stomach, or an achylia gastrica, large doses of the acid are supplied during the meal, well diluted and taken through a glass tube. This may stimulate the pancreatic secretion. **Oxyntin** capsules or tablets (combined HCl), or **acidol** tablets, may be substituted. For the treatment of the diabetes, see the article on that disease.

Surgically, **drainage** of the biliary passage is of benefit if there is persistent jaundice. The induration of the head of the pancreas may diminish.

PRACTICE OF MEDICINE—TICE

☛ Remove from Volume VII pages 1–58 inclusive and insert therefor these New Pages, 1–58 inclusive.

COURSE AND PROGNOSIS.—The disease is usually of long duration, and the prognosis depends upon the primary cause (arteriosclerosis, hepatic cirrhosis, etc.). The presence of diabetes is an important prognostic factor.

Interacinar Pancreatitis.—PATHOLOGY.—The organ is found, on examination, to be extremely tough, and there are no evidences of lobulation. Newly-formed connective tissue invades the boundaries between the cells of the acini and the islands. The connective tissue follows the supply (perivascular) and is carried close to the cells (monocellular type). The process is essentially of hematogenous or lymphogenous origin and is analogous to the portal cirrhosis of Laennec, with which it is frequently associated. The alveoli and islands may entirely disappear, due to pressure on the cells or on the blood-vessels, and nothing remain of the pancreas but a mass of fibrous tissue. This type of fibrosis predominates in the so-called bronzed diabetes, in which there is a deposit of iron-containing pigment (hemosiderin) and a result of a hemolytic process, which is accompanied by a reactive fibrosis.

HISTORICAL SUMMARY.—The pioneers in pancreatic digestive physiology were Claude Bernard, Pawlow and his Petersburg school, and Lancereaux, Mehring, and Minkowski, who demonstrated the action of the pancreas on sugar metabolism. Later Bayliss and Starling demonstrated the important action of the secretion, and overthrew the old nerve stimulus theory of Pawlow. From the clinical and pathological side Reginald Fitz of Boston was the first to draw attention to the significance of pancreatic hemorrhage. Balser first noted fat necrosis. Oser of Vienna wrote a most comprehensive article on the diseases of the pancreas and reviewed the cases reported up to that time. Korte wrote the first surgical treatise on the pancreas. Important American contributions have been made in the last decade by Opie, Flexner, and Einhorn. Robson, Cammidge, Lazarus, and Truhart have made important clinical, surgical, and pathological studies during recent years.

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CHAPTER IV

DISEASES OF THE LIVER, GALL-BLADDER AND BILIARY DUCTS

By John Ferguson, M.A., M.D.
and
W. Ewart Ferguson, M.B.

Derangements of function, p. 59—Active congestion of the liver, p. 64—Passive congestion of the liver, p. 67—Universal chronic hepatitis, p. 73—Acute non-suppurative hepatitis, p. 79—Acute suppurative hepatitis, p. 84—Chronic interstitial cirrhosis, p. 91—Hypertrophic biliary cirrhosis, p. 107—Jaundice, p. 114:—Acute yellow atrophy of the liver, p. 127; Jaundice of the newly-born, p. 134—Jaundice from phosphorus-poisoning, p. 137; Infectious jaundice—Weil's disease, p. 139. Cholecystitis, p. 145—Carcinoma of the gall-bladder, p. 155—Acute catarrhal cholangitis, p. 158—Chronic catarrhal cholangitis, p. 161—Suppurative cholangitis, p. 162—Cancer of the bile ducts and ampulla of Vater, p. 164—Abnormalities of the liver, gall-bladder and biliary ducts, p. 169—Tuberculosis of the liver, p. 174—Syphilis of the liver, p. 177—Cholelithiasis, p. 183—Hydatids of the liver, p. 196—Actinomycosis, p. 201—Cystic disease, p. 203—Benign tumors, p. 205—Malignant tumors, p. 207.

DERANGEMENTS OF FUNCTION

General considerations, p. 59—Etiology, p. 60—Functions of the liver, p. 61:—Glycogenic function of the liver, p. 61; Fat-building and fat catabolic function, p. 61; Urea-forming function, p. 62; Antitoxic or protective function, p. 62—Treatment, p. 62.

General Considerations.—The liver has a variety of very important functions to perform, the derangement of any one being a matter of much consequence to the health of the individual thus affected. It is not at all easy to state with positiveness when a process is merely a disorder of function and when it represents some organic change in the liver. Derangement of function may pass very gradually into tissue or structural changes, so that these states seem to overlap each other at many points. It is also a fact that there may be a good deal of structural change before there is any manifested functional disorder. It must also be borne in mind that there may be structural changes that are too minute to be detected, but are known to exist because of the resultant symptoms. When the symptoms are of a pronounced character there usually is such a degree of structural change as may be readily observed. On the other hand, it must be remembered that there may be very marked, almost extreme derangement of function, without the presence of any structural change in the liver. Then, further, experience has proven that there may be very extensive structural changes with but slight derangement of function and with the maintenance of health and many years of active life. The liver must be studied from its anatomical, physiological and chemical aspects.

Etiology.—Functional derangements of the liver may be primary or caused by structural changes in the organ itself; or they may be secondary to disease elsewhere. These derangements may involve only one function of the liver, or they may be general and implicate all the liver activities. Older writers, in referring to these disorders, were in the habit of speaking of biliousness, bilious headaches, torpid liver, sluggish liver, lithemia, liver dyspepsia, etc. There is considerable tendency at the present day to discount these views; but there is good ground for believing that there is much truth in them. It is now known, however, that many disorders of the liver are caused by disease or derangement of some other part of the body, and in this way it has come about that the real share of the liver in many derangements has been minimized. While it is well to keep in mind the possibilities of primary disorder in the function of the liver, it must not be forgotten that in many of the instances of lithemia the fault is not in the liver, that in most cases of disease in the bile-ducts the trouble arises primarily in the duodenum, and that in many examples of liver dyspepsia the cause is disorder in the stomach or intestines from errors in diet. The liver is, therefore, involved secondarily, and this fact should be borne in mind in considering the treatment. The amount of toxic matter produced in the intestinal canal may be greater than the liver can control or neutralize, and so give rise to serious derangement of its functions, or pass beyond it into the general blood stream. Gastro-intestinal catarrh may, in this way, cause congestion of the biliary ducts and the liver, with a certain amount of icterus. In such cases the liver is often stimulated to over-activity.

There are conditions where there are reasons for regarding the liver as under-active, such as the chronic congestion of the organ in cardiac failure and in old-standing pulmonary disease. In these cases there is usually a defective state of elimination and some of the symptoms may arise in this way. But some of the symptoms are also due to the fact that the imperfect functioning of the liver permits toxic matter to pass through it and thus reach the general circulation, causing the headaches and other symptoms that have often been attributed directly to some faulty action of the liver.

It should be remembered that there are cases where the poison or irritant reaching the liver is so virulent or in such amount as to destroy much of the parenchyma of the organ. A very complicated state of functional failure will result, revealed by disturbances in the biliary, glycogenic, urea-forming, fat-storing, and toxin-destroying properties of the gland. In such cases there may be marked headache, active delirium, extreme nervousness, pronounced jaundice, and, finally, stupor or coma. Such a combination of symptoms is met with in acute yellow atrophy.

Functions of the Liver.—The functions of the liver may be summarized as follows: the secretion of bile; the storing of fat taken from the food, to be set free as required; the formation of glycogen from the carbohydrates of the food and certain constituents in proteins, and reconverting the glycogen into glucose as required for the nutrition of the body; the metabolization of proteins and the formation of urea; the destruction of red blood-corpuscles; and the rendering inert the poisons brought to it from the intestinal canal or other parts of the body.

Glycogenic Function of the Liver.—The glycogenic function of the liver has been the subject of much discussion. Claude Bernard held that the liver formed this substance and again converted it into glucose, which entered the blood by way of the hepatic vein. Pavy, on the other hand, held that the liver during life did not convert glycogen into glucose, but produced fat and protein from it. According to Halliburton, the prevalent opinion among the leading physiologists is that the liver forms glycogen from carbohydrates mainly, and to a much lesser extent from proteins; that part of this store is converted into fat; that part unites with protein to form a glucoprotein; but that most of the glycogen leaves the liver as glucose, or what is usually designated as sugar. It has been observed that the arrest of the flow of the bile lessens the power of the liver to store glycogen, though not its power to form it. From this it has been concluded that a hepatic patient would be likely to suffer from glycosuria. This has been established clinically by observing that nearly all cases of hepatic colic have a concurrent glycosuria, due to the formation of glycogen and the failure of the liver to act as a storehouse for it. Careful study has revealed a difference in the metabolism of dextrose and levulose, the former being converted into glycogen by the muscles as well as by the liver, while the latter is converted into glycogen almost solely by the liver. In hepatic cases there is, therefore, a low tolerance for levulose, and a much higher tolerance for dextrose, as the muscle acts in a compensatory capacity.

Fat-building and Fat-catabolic Functions.—Much attention has been devoted to the fat-building and fat-catabolic functions of the liver. Leathes, Hartley, MacLeod, and others have shown that the power of building up fats, such as the phosphatids, is essential in the formation of certain tissues. But these authors have also shown that the liver takes an important part in the preparation of fats for their final splitting up into carbonic acid and water. The lipase stored in the connective tissue sets free the fatty acids and glycerol. These are then transported by the blood to the liver, where further changes occur. In cases of a deficiency of glycogen, either from starvation or from excessive loss of sugar, as in diabetes, the breaking up of fats in the liver may become very rapid, causing acetones, diacetic, caproic, and beta-oxybutyric acids to appear in the blood, giving rise to acidosis. Butyric acid and beta-oxybutyric acid are probably normal products in fat catabolism, but in health and on a normal diet these are further oxidized into carbonic acid and water. When carbohydrates are withdrawn from the diet the fat-splitting is arrested, mainly at the hydroxybutyric acid stage.

Urea-forming Function.—The urea-forming function of the liver is one of the most important in the animal economy. A small portion of the amino-acid group is used in tissue or endogenous metabolism. The larger part of the nitrogenous portion of the food is converted into urea by the liver. When the liver is removed, urea almost disappears from the urine and ammonia takes its place. When the portal vein is connected with the hepatic vein (Eck's fistula), very little urea is found in the urine, and ammonia makes its appearance. In cirrhosis and acute yellow atrophy of the liver there is little urea excreted, while the ammonia is much increased. In this latter disease such amino-acids as leucin and tyrosin are found in the urine, caused by the disintegration of liver cells. If the amino-acids, such as glycin, leucin, etc., are injected into the blood or fed to an animal, urea will at once be excreted in excess in the urine.

Antitoxic or Protective Function.—The antitoxic or protective function of the liver calls for notice. This organ exerts a great influence on poisons brought to it from the intestinal canal, whether these are introduced into the canal from without, or are produced there by bacteria. The influence of the liver is shown when a poison is injected into the portal vein or into the general circulation, the result being much less harmful by the former than by the latter route. Roger, Gley, Buchard, Gueirolo, Senator, and others have devoted much study to this function of the liver. It is now very generally admitted that this organ has in a marked degree the power to oxidize and render many poisons inert or less virulent. It is also known that the more perfect the glycogenic function of the liver, the more energetic is the antitoxic power of the organ. When the liver loses the power of converting sugar into glycogen it also loses its antitoxic function. This is also true in biliary obstruction—a condition in which the system soon becomes flooded with toxins.

Treatment.—General Management.—In the treatment of the functional derangements of the liver it is necessary to **consider the cause.** Inquiry into the occupation, mode of life, diet, habits, state of digestion, and action of the bowels will reveal the direction that should be followed in the treatment of these cases. In many persons complaining of what they call liver trouble there will often be found present some irregularity in diet and digestion, constipation or gastro-intestinal catarrh. The keynote in treatment is **regulation** and **elimination.**

Diet.—The first thing to consider is the diet. It may be found that the victim of liver trouble consumes too much fat, sugar, starch, or protein. Guidance as to the proper quantities of these constituents of one's diet must be laid down. Directions as to the time of eating and the total amount to be consumed are also necessary. The best efforts at treatment may fail if these fundamental principles are overlooked. Much dyspepsia and secondary liver trouble arise from defective mastication. This throws an extra quota of work upon the digestive organs that ends in irritation and, finally, in confirmed disease. **Proper cooking and preparation of the food** is of the utmost importance. Choose a simple diet and avoid a rich one is the master word; and during symptoms adopt a liquid dietary.

Beverages.—As to the use of beverages careful rules must be laid down. During active biliary symptoms, alcoholic drinks, especially ale, beer, stout and porter, should be avoided. Cider and the heavier wines are also objectionable, such as port, madeira, Burgundy and champagne. If any alcoholic beverages should be required, claret and a moderate quantity of whiskey and water are the best. Among drinks, **water** takes first place. It is the best of all solvents and carries away, through the skin and kidneys, much toxic matter. It also aids greatly the action of the aperients administered to regulate the bowels. Water may be taken hot before breakfast and on going to bed. It may also be imbibed freely a short time before taking food. As a rule, it is not well to drink water too freely while at meals. On this point, however, experience must be the real guide. One dyspeptic with pronounced biliary symptoms was cured by consuming large quantities of water at meal. times. The merit of the spa and medicinal water treatment lies in the amount of water, rather than the salines that are consumed. In the vast majority of cases these can be made up at home. One of the results of drinking large quantities of water is to raise the portal vein blood-pressure. This causes a freer circulation through the liver and in turn a freer flow of bile of more liquid composition and under higher pressure in the biliary tubes. In this way both the venous and biliary systems of the liver are "flushed."

Exercise.—Exercise in keeping with the strength, habits and age of the patient should be enjoined. At first it may have to be of a simple and gentle character, and may have to be of some indoor sort. Gymnastics are often valuable and various forms of movement cures, such as the Swedish, have filled a useful place. **Walking, rowing, golfing, and the saddle** are among the best forms of exercise. As an exercise calculated to stimulate digestion, and particularly the liver, none can surpass **horseback riding.** A **Turkish bath** with a good rubbing to rouse the action of the skin is a therapeutic measure that should not be lost sight of.

MEDICINAL TREATMENT.—The important thing in drug treatment is to keep in mind the fact that the bowels must be regulated, and suitable aperients take first place. Of these none fill a more useful rôle than **calomel followed by a saline.** The usual dose of calomel is 3 grains (0.2 gram), but some prefer the ordinary blue pill in 5-grain (0.325

gram) doses. In some cases of chronic intestinal indigestion, with accompanying liver symptoms, small doses of calomel, 1/20 grain (.00325 gram), three times a day, prove very valuable. There are some cases of intestinal catarrh—the result of errors in diet—that are greatly relieved by daily teaspoonful doses of castor oil taken in the morning. These drugs are much more useful than the so-called intestinal antiseptics, the efficacy of which may with good reason be doubted. For the purpose of emptying the portal circulation and ameliorating the symptoms of the "torpid" liver, few combinations are better than the **compound cathartic pill,** now altogether too infrequently employed. **Salicylate of soda** is an excellent liver stimulant.

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ACTIVE CONGESTION OF THE LIVER

(*Arterial Congestion, Active Portal Congestion*)

Etiology, p. 64—Symptomatology, p. 65—Diagnosis, p. 65—Treatment, p. 65—Prognosis, p. 66—Pathology, p. 66.

Etiology.—In studying the etiology of active congestion of the liver it is necessary to bear in mind that the parenchyma of the organ is very fragile, that the vessels are very numerous, and that the capillaries possess very thin walls. These factors enable the liver to act as a reservoir for blood and to permit of marked variations in the amount which at any time it may contain. A rise in the blood-pressure in the vena cava will raise the pressure in the hepatic vein and slow down the flow from the liver. On the other hand, a rise in the blood-pressure in the hepatic artery or the portal vein will cause more blood to flow into it. Among the causes that induce liver congestion, alcoholic and dietary excesses take a first place. The passing to the liver of many poisons from the intestinal canal or from the general blood stream is also a potent cause. Residence in hot climates has been given as a cause on rather doubtful evidence, similarly cold and chills, as urged by Cantlie, have been set up as causes, though proof is scanty. Many infections, as typhoid fever, yellow fever, influenza, malaria, and tropical dysentery, certainly give rise to the condition. Chronic congestion, former attacks of malaria, and sedentary habits act as predisposing factors. Congestion of the liver may be caused by injuries;

and it is claimed by many authors to be due to the arrest of blood loss, as in the cessation of the menses or hemorrhages from piles.

Symptomatology.—The symptoms of active congestion of the liver are usually distinct and readily recognized. The condition is that often called a "bilious attack." There is the history of some indiscretion in diet or the abuse of alcoholic beverages. By these means the blood becomes loaded with toxic material, and the channels of elimination are more or less inactive. There is loss of appetite, and the thought or sight of food creates nausea or disgust. The tongue is coated, there is a bad taste in the mouth, and distress or pain in the epigastrium, and not infrequently there is vomiting. All these symptoms are aggravated by the taking of food. There is usually some pain in the region of the liver, which is increased on moving or lying on the side. There is also tenderness on pressure. Usually there is constipation, but the errors in diet that induced the attack may cause diarrhea. The nervous symptoms are generally well marked, and consist of headache, vertigo, malaise, poor sleep, irritability, depression, muscæ volitantes, and an inability to concentrate thought upon work.

PHYSICAL FINDINGS.—There is some enlargement of the liver, which can be felt beyond the false ribs on the right side. The urine is reduced in amount, is of high specific gravity, is dark in color from the presence of urobilin, and contains an increased amount of urates. There may be a slight rise of temperature of one or two degrees, due to a moderate hepatitis. This is likely to be the case when the liver disturbance follows an attack of dysentery. When jaundice is present it is usually slight in degree as compared with that caused by obstruction of the biliary tubes or when it is of catarrhal origin. The stools may be lightish in color, due to a suppression of the secretion of bile, though generally they are of normal appearance.

Diagnosis.—Active congestion of the liver may be distinguished from *perihepatitis* by the absence, in the latter, of a history of errors of diet or excess in drinking, and also by the absence of any enlargement. In perihepatitis there is usually a history of syphilis. The pain and jaundice of active congestion of the liver may resemble sufficiently closely similar symptoms found in some cases of right-sided *pleuropneumonia,* and care should be taken to avoid an error in diagnosis.

Treatment.—GENERAL MANAGEMENT.—The first thing to consider in the treatment of active congestion of the liver is the *causation* of the attacks.

Diet.—All errors in diet and the use of alcoholic beverages must be corrected. Only small amounts of meat should be allowed and then only of some tender and soft kind. A **milk and vegetable diet** meets the requirements of most cases. A variety of vegetables and farinaceous foods may be tried, and the wishes of the patient may be followed, to some extent. Fat, cured and pickled meats, alcohol and strong spices must be excluded. Whey, junket, albumin water, thin broth, barley water, calve's foot jelly, etc., are among the nutriments that may be first tried. Milk, well diluted with lime water or some effervescent water, may be borne at an early period in the illness. As the attack passes off the diet may be gradually increased in amount and variety.

Local Treatment.—The insertion of a hollow needle three and one-half inches in length into the liver, with the object of **drawing off blood,** has been practiced by Cantlie. The needle may have to be inserted several times before a vein of sufficient size is punctured to remove an amount of blood sufficient to relieve the congestion. The risk of bleeding into the peritoneal cavity, or of striking the vena cava, would be ample to deter most physicians from trying this line of treatment. The application of **leeches** around the anus has been advised.

The prevention of future recurrences by a **strict regulation of the habits of life** must receive due consideration. Directions must also be given regarding the **clothing,** so as to avoid chills.

MEDICINAL TREATMENT.—The medicinal treatment should, in the early period of an attack, be limited to **saline cathartics,** such as the laxative mineral waters, Seidlitz powders, or magnesium sulphate. At this stage the stimulating aperients such as calomel, podophyllin, rhubarb, blue mass, jalap, colocynth, etc., should not be ordered, as they tend to increase the congestion of the liver. Later in the disease they are of distinct value. Murchison recommended **ammonium chlorid** in doses of 20 grains (1.3 grams) three times a day. This drug may be administered at any time during the illness. Murchison gave it to produce free diaphoresis and reduce the congestion of the liver. **Ipecacuanha** has had considerable reputation in India. It is given in doses of 20 grains (1.3 grams) every six to twelve hours, preceded about half an hour by ½ grain (0.0325 gram) of opium to prevent vomiting. For the gastro-intestinal symptoms **alkalis, the alkaline waters, carbonate of magnesia, bismuth, and hydrocyanic acid** are useful, and recommended by Savill. When the irritation has subsided, mild, bitter tonics of **nux vomica, gentian calisaya, etc.,** are helpful in regaining the appetite. If the congestion becomes chronic no drug appears to yield as good results as 10-20 minims (0.6-1.25 c.c.) of the dilute **nitrohydrochloric acid.** This should be combined with **quinin** in malarial cases. **Local hot or cold packs** will relieve both the congestion and the pain.

Prognosis.—This should be given somewhat guardedly, as the congestion may be the forerunner of some serious affection. As the subject is apt to continue his dietetic indiscretions, the attacks are prone to recur at shortening intervals and with increasing severity, ending in chronic congestion or cirrhosis. An ordinary attack rarely lasts more than one week.

Pathology.—The liver presents the appearance of a congested organ, as it is enlarged, swollen, and contains an increased quantity of blood, giving it a dark-reddish or reddish-brown color. It drips blood on section and the capillaries and small vessels are seen dilated. There is sometimes degeneration in the parenchyma and there may also be fatty degeneration. Pigmentation of the liver cells is also found in some cases. Swelling, congestion, and proliferation of the biliary ducts have occasionally been met with. Chronic recurrent cases may cause induration of the liver, or true cirrhosis.

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PASSIVE CONGESTION OF THE LIVER

(Nutmeg Liver, Cyanotic Atrophy, Cardiac Liver, Hepatic Asystole, Venous Congestion, Red Atrophy, Passive Hyperemia)

Etiology, p. 67—Symptomatology, p. 67—Diagnosis, p. 69—Complications and sequelæ, p. 70—Treatment, p. 70:—Prophylaxis, p. 70; General management, p. 70—Prognosis, p. 71—Pathology, p. 72.

Etiology.—The etiology of passive hyperemia of the liver may be stated as follows: cardiac insufficiency arising from valvular disease; impairment in the myocardium, or pericardial lesions; some morbid condition in the lungs, as asthma, chronic bronchitis, emphysema, thickened and adherent pleuræ; stasis in the blood-flow in the inferior vena cava due to tumor, aneurysm, spinal deformity; the congestion accompanying pneumonia, typhoid fever, and other infections. This is a temporary form and does not give rise to permanent damage to the organ.

Symptomatology.—Clinical History.—*Mode of Onset.*—The onset of passive congestion of the liver is gradual and the engorgement and morbid changes in the liver steadily advance, in keeping with the morbid conditions that hold the relationship of causation. As the obstruction to the return of the blood from the liver continues and becomes more and more marked, the changes in the liver increase until its several functions are seriously impaired. There soon develops a feeling of tightness, fullness, and discomfort in the region of the right lobe of the liver. Dyspnea, weakness, and inability to work soon appear.

Symptoms During Progress of Disease.—These are both objective and subjective, but as the former will be taken up fully under Physical Examination, we will mention them only in passing here, together with some subjective symptoms. The patient suffers from pain in the liver, which may be due to an accompanying perihepatitis. There is some loss of appetite which may become very pronounced. The usual symptoms of dyspepsia, such as flatulence, constipation, diarrhea, and abdominal distention, are often present. Dyspnea is a more or less constant condition, and is due to the cardiac or pulmonary disease that constitutes the primary malady. As the disease advances, insomnia may become the source of much distress. Nausea is usually present, and vomiting is not infrequent; the vomit may occasionally contain blood. The patient may be conscious of a pulsation in the liver. There is frequently considerable distress after taking food, owing to the turgid state of the liver, and to the fact that eating increases the vascularity of the gland.

PHYSICAL FINDINGS.—(*a*) *Observation.*—The facial expression is often quite characteristic. The skin over the malar eminences is congested and of a high color. The lips and ears tend to be cyanosed. The skin over the forehead and temples has a slightly icteric tint. The contrast between these portions of the skin is readily seen and is distinctive of the advanced case. Pulsation may be visible in the veins of the neck and in the liver, caused by waves of blood thrown into the venous system by the cardiac systoles. There may be a certain fullness visible in the right hypochondrium, the abdomen may be distended with fluid, and edema of the hands and legs can usually be seen, especially in the advanced stage of the disease. As the disease advances jaundice may become moderately developed, due to pressure on the biliary tubules; and this, combined with the cyanosis of the skin, produces a greenish color.

(*b*) *Palpation.*—The edge of the liver may be felt to extend for two or three fingers' breadth below the last rib. Considerable tenderness may be elicited by pressure, which is either superficial in the skin, extending from the seventh to the tenth dorsal vertebræ, or deeply seated in the liver. Pulsation in the liver may be felt and is synchronous with cardiac systole. It is detected by placing one hand over the ensiform cartilage and the other over the right side at the margin of the ribs. It is a heaving, diffuse impulse, and must be distinguished from the communicated throb of the diseased heart. By pressure upon the liver in an upward direction considerable impediment to the flow of blood in the inferior vena cava may be produced, causing marked blood waves to appear in the jugulars. The edge of the liver is firmer than normal and somewhat rounded, and the surface is smooth and even in contour, differing from that found in cancer, cirrhosis, and hydatids. Palpation may reveal the presence of anasarca.

(*c*) *Percussion.*—Percussion confirms what has already been made out by observation and palpation. An increased area of dulness is found to exist in the liver region. This may extend upward, the swollen liver pressing the diaphragm against the right lung. The dull area extends also toward the left hypochondrium, and may descend below the umbilicus. By percussion, fluid may be discovered in the abdominal cavity, and the size and position of the heart can be mapped out and also the condition of the lungs ascertained. The thorough employment of percussion may reveal the presence of a new growth or an aneurysm in the thoracic cavity as the cause of the obstructed blood flow in the inferior vena cava.

(*d*) *Auscultation.*—The heart and lungs should be carefully auscultated. This enables one to form some opinion as to the degree of advancement of disease in these organs. By this means it may be possible to determine the presence or absence of a thoracic aneurysm. The kind of valvular lesion can also, in this way, be detected. When hepatic pulsation is well marked, a liver bruit has sometimes been heard.

LABORATORY FINDINGS.—The laboratory findings in passive hyperemia of the liver are rather indefinite and inconstant. The vomit and feces may contain blood, and sometimes the latter are clay-colored if jaundice is present. The urine is usually scanty, and, in advanced cases, of high specific gravity, and high color, due to the presence of urobilin, with abundance of urates. As the disease progresses there is an increasing tendency toward the appearance of albumin in the urine due to a congested state of the kidneys which may impair the renal epithelium. Glycosuria is very rarely present unless there is disease of the pancreas. The daily amount of urea is an index of the hepatic action; and when reduced in amount the liver is correlatively functionating insufficiently.

Diagnosis.—A careful perusal of what has been said regarding the symptomatology and physical and laboratory findings will form the basis for a diagnosis. There is the presence of an enlarged and tender liver with organic disease of the heart. The volume of the liver in passive congestion is liable to marked variations, whereas the liver that is enlarged from other causes remains constant in size. The exhibition of cardiac tonics or a brisk purgative rapidly reduces the size of a congested liver.

In *cirrhosis of the liver* there are enlarged abdominal veins, an absence of cardiac disease, the absence of pain and tenderness, the appearance of ascites before anasarca, and the occurrence of hematemesis. The smooth surface, the variableness in size, and the absence of cachexia will distinguish the congested liver from the malignant one. The absence of chronic suppuration will exclude *amyloid disease*, and an examination of the blood will decide the case if *leukemia* is suspected. The history of the case, with special tests, will clear up any doubt as to the condition being due to *syphilis*.

In a case where there are ascites, anasarca, enlargement of the liver, and albuminuria, the question arises whether the heart, the liver, or the kidney is the primary seat of disease. If the anasarca preceded the ascites it is evidence of a cardiac origin; and, as enlargement of the liver rarely follows renal disease, the latter may be excluded as the primary source of the syndrome mentioned. On the other hand, the enlargement of the liver is a fairly constant result of *valvular dis-*

An *x*-ray plate or the careful screening of the patient will aid in completing the diagnosis, by revealing the relative size and position of the heart, liver and spleen.

Complications and Sequelæ.—As the disordered condition of the liver advances *pari passu* with the cardiac or pulmonary diseases from which it has sprung, several complications may arise. Hematemesis is likely to occur in about 25 per cent. of the cases, caused by the congested state of the stomach. This condition may extend into the duodenum and produce much gastro-intestinal disturbance, such as flatulence, constipation and, less frequently, diarrhea. Arising from these derangements there may be considerable auto-intoxication, and an extension of the duodenal congestion into the bile-ducts, followed by jaundice. Jaundice may also be caused by pressure upon the fine biliary tubules in the liver by the distended veins—a form of jaundice that may become very severe, a true icterus gravis—from atrophy of the parenchyma. The backward pressure in the portal circulation causes some ascites in most cases and, at times, a very large amount of fluid may accumulate in the abdominal cavity. From this accumulation of fluid and impaired circulation peritonitis and perihepatitis have resulted. Most writers state that the spleen may become enlarged, but Quincke is of the opinion that it does not show enlargement as it does in cirrhosis of the liver, but, on the contrary, is indurated. Due to the impaired state of the general circulation, as well as that of the abdominal cavity, the kidneys may become congested and albuminuria become more or less constant. In time the liver becomes cirrhotic, or rather a condition of hepatic fibrosis ensues. The liver ultimately may lose its power of destroying toxic products to such an extent that a condition of stupor, delirium and coma may result from auto-intoxication.

Treatment.—PROPHYLAXIS.—Something can be done for those who have cardiac disease, tumors pressing upon the thoracic portion of the inferior vena cava, or emphysema, that may materially lessen the frequency and severity of the recurrent attacks of hepatic hyperemia, or, perhaps, prevent their advent in such cases. By the careful **regulation of the diet, the occupation, and the habits** of such persons, much can be accomplished for their comfort and the prolonging of life. Not a few who would eventually undergo all the sufferings of hepatic hyperemia, may, by judicious prophylactic management, escape this hyperemia, and have their ailments limited, to a great extent, to the primary disease. A strenuous and laborious life, together with every form of excess, must be forbidden; while abstemious habits with regard to alcohol, the adoption of a simple diet, and regularity in all things are enjoined. Medicinal treatment for the heart and symptoms may do much in the way of prevention.

GENERAL MANAGEMENT.—*Diet.*—The diet should be plain and light, consisting mainly of milk, vegetables, cereals, weak soups, and the **avoidance of all forms of alcohol.** The food must be nutritious, easily digested, and not too bulky, and **fluids should be as small in amount as**

possible. This latter point is very important, as many patients begin to improve upon restriction in the consumption of liquids.

Local Treatment.—**Hot applications** over the liver have been recommended. Harley and Cantlie have recommended and practiced the **withdrawal of blood** from the liver through a hollow needle. **Leeches** have been applied over the liver and around the anus. **Bleeding from hemorrhoids** should be encouraged. The writer has found **venesection** from the arm a very valuable means of promptly relieving the heart, the swollen liver, and dyspnea. **Tapping** may be required for the ascites, and **puncture** or the use of **Southey's tubes** for the edema.

Treatment of Symptoms.—Pain often calls for relief, and for this purpose hot applications are useful. **Morphia** in small doses should be administered hypodermically, grain 1/10 (0.006 gram). Small doses are quite effective for the *relief of the pain* and less disturbing than the usual dose of grain ¼ (0.016 gram). *Insomnia* may be troublesome. The favorite drugs are **trional, veronal, sulphonal, chloralamid, paraldehyd,** some form of **opium,** and the **bromids. Paraldehyd** in 1 dram (4 c.c.) doses with half an ounce of **whiskey** is a trustworthy hypnotic. For most cases **veronal** in doses of 5-10 grains (0.325-0.65 gram) should be given the preference.

Medicinal Treatment.—The chief drugs upon which one must rely are those that act favorably on the primary disease, and such as relieve the condition of the liver and portal circulation. One of the most helpful lines of treatment is a brisk saline purgative, and for this purpose a saturated solution of **magnesium sulphate** is the best. The action of the saline can be increased by administering such cathartics as **podophyllin, rhubarb, colocynth, jalap, elaterium, or aloes.** An excellent remedy for the *anasarca, ascites, and cardiac condition* is **Addison's pill,** known as **Guy's hospital pill,** composed of 1 grain (0.065 gram) each of digitalis, squills, and blue mass. This may be given three times a day. For the *regulation and toning of the heart,* **digitalis** is the most reliable drug and may be given in the form of the powdered leaf, 1 grain (0.065 gram) every four to six hours, or of the infusion, or a good tincture in doses of 10-20 minims (0.6-1.25 c.c.) three times a day. **Caffein,** grains 1-5 (0.065-0.325 gram); **strychnia,** grain 1/64-1/20 (0.001-0.003 gram); and **camphor,** grains 2-5 (0.13-0.325 gram), have been recommended as cardiac tonics. When there is reason to believe that there is impairment in the heart-muscle, the writer knows of no drug equal to **arsenic,** which may be given in the form of arsenious acid, grain 1/20 (0.003 gram), three times a day after food. Diuretics are of very doubtful value, but **theobromin,** and **salicylate of soda,** grains 5-30 (0.325-2 grams), **spartein sulphate,** grains 1/12-2 (0.005-0.13 gram), and **potassium citrate,** grains 15-30 (1-2 grams), every four hours, may be tried.

Prognosis.—As the great majority of cases of hepatic passive hyperemia are caused by incurable organic heart disease, or some serious lung malady, or the presence of an aneurysm or a tumor in the thoracic cavity, it will at once appear that there is little hope for a cure of the liver condition. As the primary diseases are usually permanent and fatal, the hyperemia must continue to the end of life, except when caused by typhoid fever, pneumonia, or some other infection. There is at times some improvement corresponding with whatever improvement may be effected in the organs primarily involved, especially the heart; but these periods of improvement are always followed by relapses. When once pulsation has occurred in the liver it never again disappears. The mode of death in most of the cases is that of progressive heart-failure; but there may be a terminal infection of some part of the body, as the lungs, the kidneys, or the liver itself. When the liver becomes infected there is usually vomiting and internal hemorrhage. A grave type of jaundice develops and the patient passes into a state of low delirium, with stupor and coma. The prognosis as to the functional capacity of the liver is largely one of the maintenance of the general circulation and especially the degree to which the heart may continue to perform its functions. So, therefore, the outlook regarding the liver in the performance of its duty in the body economy is equally as gloomy as that for its chances of a recovery. Recovery of function takes place when the hyperemia is caused by some infection, as typhoid fever.

Pathology.—POSTMORTEM FINDINGS.—When the liver is examined postmortem, it varies much in size, appearance and color with the duration of the illness, the extent of the congestion, and the time after death when the body is opened, as the organ shrinks in size soon after death, and, with this reduction in turgescence, it tends to become paler in color. When death occurs in the early period of the disease, the liver is enlarged, its borders are thickened, its substance is firmer than normal, its surface will indent with finger pressure, and the color is dark brown or bluish-purple. When cut, blood drips from the enlarged hepatic veins. The cut surface presents dark brown areas surrounded by a margin of lighter color, an appearance which resembles a cut nutmeg —hence the name nutmeg liver. The dark areas are those surrounding the hepatic or intralobular venules, while the lighter areas are those supplied by the compressed portal or interlobular venules, where bile and fatty matter have been deposited. At a later stage in the disease, the liver may appear to be smaller, due to the formation of contracting fibrous tissue, and the atrophy of the cell elements, and the condition called red atrophy appears, though the nutmeg characteristic still remains. On section the liver shows a tendency to granulation, due to the projection of lobules above the surface, caused by the contraction of the connective tissue. The serous covering of the liver may be thickened, and there may be adhesions between it and the serous surface of the diaphragm.

MICROSCOPICAL FINDINGS.—The microscope reveals some very important changes. The intralobular venules and capillaries are dilated. and the centers of the lobules have a mahogany-red color, due to an extra amount of blood, and the infiltration of the cells with pigment and blood-crystals, from capillary hemorrhages. The portal venules and capillaries are found to be compressed by the swollen lobules, causing a relative anemia in the interlobular areas. This arrested flow of blood in the portal capillaries leads to the deposit of a considerable quantity of the fatty matter brought from intestinal absorption, a condition that has been called the fatty nutmeg liver. In addition to these changes there is more or less stasis in the biliary capillaries and the deposit of bile coloring matter in the interlobular spaces. Together these deposits create the lighter or grayish margins that surround the dark lobular centers. According to the stage and duration of the hyperemia, the amount of cellular atrophy will vary. In like manner the degree of fibrosing will be subject to marked differences in extent. This fibrosis proceeds from both the hepatic and the portal veins, and the lobule is thus invaded from its center and its periphery.

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UNIVERSAL CHRONIC PERIHEPATITIS

(*Perihepatitis Chronica Hyperplastica, Sugar-iced Liver, Chronic Deforming Perihepatitis, Chronic Hyaline Perihepatitis, Capsular Cirrhosis, Multiple Serositis, Polyorrhomenitis*)

Etiology, p. 73—Symptomatology, p. 74—Diagnosis, p. 75—Association with other diseases, p. 76—Treatment, p. 76:—Prophylaxis, p. 76—Prognosis, p. 77—Pathology, p. 77.

Etiology.—The etiology is quite obscure, and most of what has been given by various writers is purely speculative. Hale White claims that a *microörganism* is the cause of the trouble. Again, others state that it is due to *Bacillus tuberculosis*. To this the author cannot agree, as the morbid changes found do not in any way correspond to those found in tuberculosis; and, further, tuberculosis is very common, while general chronic perihepatitis is very rare.

In a very large percentage of the cases there is arteriosclerosis and granular red kidneys. But these changes cannot be set down as the cause; for the chronic red kidney with hardened arteries is seldom found to give rise to perihepatitis, and these cases of chronic Bright's disease escape this complication. It is not a sequel to interstitial nephritis.

Then, further, syphilis has been advanced as an etiological factor. But it should be borne in mind that syphilis is common while perihepatitis fibrosa is not. The lesions found in the disease are not those of syphilis.

In like manner gout, rheumatism and malaria cannot be made to answer the demands for a true etiology.

All the evidences point, however, to some infection of persistent though mild action, leading to organization of cicatricial connective tissue. The infections and diseases mentioned may stand in a coöperative sense as causes. They may lower resistance and, in this way, aid the causative organism. Arteriosclerosis and granular contracted kidneys seem to stand in this relationship more frequently than any other condition. Thus it comes to pass that though there are not many cases of fibrous perihepatitis, these few are nearly always victims of an arteriorenal pathology.

The disease is equally common to both sexes. With regard to age, it may occur from 20 to 70 years of age, and the average has been found to be about 43 years. Heredity does not influence the disease.

Judging by what is known by bacteriology, the great weight of evidence favors the view that it is a type of streptococcic infection; and this is the view of the writer after much study and considerable experience with the disease.

The theories that have been mentioned by some regarding trauma, tight lacing, alcohol, etc., would, therefore, only be predisposing causes.

Symptomatology.—CLINICAL HISTORY.—*Mode of Onset.*—The mode of onset is slow, the disease having perhaps lasted for years prior to the patient's knowledge that any serious malady was present, until adhesions to adjacent structures—as the diaphragm and abdominal walls—or decided thickening of the serous covering of the liver, appear.

Symptoms During Progress of Disease.—The main *objective sign* of the disease is the occurrence of ascites, which always appears and distends the abdomen. There may be edema of the feet and legs from renal or cardiac disease, or from pressure on the vena cava by the thickened peritoneum.

The *subjective symptoms* are not definite, are often quite vague, or there may be none. Occasionally there is pain, but more frequently there is only a feeling of discomfort in the upper portion of the abdomen, such as fullness and weight. At times there is a sudden onset of pain, probably caused by an inflammatory process. The abdominal walls may become painful from distention. General health is usually well maintained, and there is freedom from gastro-intestinal derangements in most

Physical Findings.—*Observation.*—The most noticeable thing about this disease is the distention of the abdomen with fluid. Very rarely is jaundice observable. Edema of the feet and legs is sometimes present. The heart may be seen to be irregular and with a wide and forcible impulse against the chest-wall. The breathing may be disturbed through accumulations of fluid in the pleural cavities.

Palpation.—By palpation the edge of the liver may be found to be thickened and tender. Sometimes the contracted omentum may be rolled up and felt as a ridge which must not be confused with the border of the liver. Friction can sometimes be felt in the right hypochondrium. When the liver is palpable it is smooth, but may feel more rounded than normal. When the disease has become advanced, the liver, though smooth, is frequently felt to be more or less distorted in shape. If edema is present pressure produces pitting.

Percussion.—Percussion will reveal the presence of fluid in the abdomen, or in the pleural cavities. Sometimes there is pericarditis with an enlarged area of cardiac dullness. The spleen may be found enlarged, but this is not usual, as its thickened capsule acts as a preventive in this regard.

Auscultation.—Auscultation may discover the presence of heart disease or pleurisy, both of which are common accompaniments of chronic perihepatitis.

Laboratory Findings.—Albuminuria is present in a considerable number of the cases, for the reason that interstitial nephritis is frequently in association with this liver disease. The ascitic fluid is clear and straw-colored, but when it stands for some time threads of fibrin may form. It also contains about 3 per cent. of albumin, which points to the fact that the exudate is due to some inflammatory process and not merely a transudation.

Diagnosis.—The diagnosis of perihepatitis is often very difficult to make. So eminent an authority as Osler states that he has not been able to decide definitely between this condition and *cirrhosis of the liver with ascites.* According to Hale White, the three chief points in diagnosis are interstitial nephritis, recurring ascites, and the absence of jaundice. There is a group of cases that are associated with pericarditis, generally adherent, and inflammation in the mediastinum.

The most remarkable characteristic of this form of chronic perihepatitis is the tendency to recurrent ascites, which lasts for many years, and requires tapping many times. Osler mentioned the case of a patient on whom this operation was performed 121 times, and Rumpf a case on whom it was performed 301 times. Between the tappings there are often periods of fairly good health, when the person may be able to attend to his usual vocation. It is difficult to differentiate between perihepatitis and true cirrhosis of the liver. If jaundice occurs the probabilities are in favor of cirrhosis. In cirrhosis the fluid forms more rapidly and the patient very seldom lives to require a second tapping, as life is nearing its end by the time this treatment becomes necessary.

It is also necessary to distinguish chronic hyperplastic perihepatitis from *perihepatitis, acute or chronic,* caused by abscess in the liver, the presence of tubercle or cancer, or the extension of an inflammatory process from an adjoining part, such as an empyema. None of these forms of perihepatitis run the long course of the form now discussed with many remissions and tappings. In one case the total fluid removed was 790 pints, and in another it was as much as 2,000 pints.

As a special aid in effecting a diagnosis must be mentioned the aspirating needle, which should be inserted in the seventh or eighth intercostal space in the midaxillary line. If the fluid is ejected on inspiration it comes from beneath the diaphragm, and if it is ejected on expiration it comes from the pleural sac. The nature of the fluid thus secured is of importance, the empyematous being purulent, while that from perihepatitis is clear and rich in albumin.

Association with Other Diseases.—This affection of the serous covering of the liver is often associated with some other disease, such as pleurisy, empyema, mediastinitis, adherent pericarditis, chronic nephritis, gout, rheumatism, syphilis, tuberculosis and arteriosclerosis. Some of these conditions may be merely accompaniments, at other times some of them may be results, or, again, they may be the causes of the perihepatitis. This latter is specially true with regard to syphilis, tuberculosis, and interstitial nephritis.

Treatment.—PROPHYLAXIS.—But little can be done for the prevention of this condition, except in the way of treating such diseases as tuberculosis, gout, syphilis, pericarditis, and nephritis, should these be found to exist.

DIET.—This should be of the variety that is easily digested, nutritious, and such as agrees best with the patient. Stimulating and rich foods and such articles as cause flatulence or other symptoms of indigestion must be avoided. Liquids should be carefully limited, and also salt.

MEDICINAL TREATMENT.—Outside of such drugs as may be required for the relief of symptoms such as pain, sleeplessness, debility, or constipation, there is little that is specific apart from **quinin,** in cases with a prior malarial history, and the proper treatment for syphilis when the history and the blood-test are definite. For this latter disease **potassium iodid** may be prescribed in doses of 5-15 grains (0.325-1 gram) three times daily; or a **mercurial** such as the **protiodid,** grain 1/6 to grain ½ (0.01-0.03 gram). A mixed mercurial and iodid treatment may be adopted, such as potassium iodid, grains 5-15 (0.325-1 gram), and **mercury perchlorid,** grain 1/64 to grain 1/10 (0.001-0.006 gram) in solution after meals. The intravenous treatment with some preparation of **arsenic** may be employed.

Much of the drug treatment is to be directed toward the accompanying maladies. Diuretics have been tried with doubtful results, the favorite ones being **caffein, strophanthus, digitalis, diuretin,** and **squills.** Hale White regards **copaiba resin** as the best diuretic, in doses of 15 grains (1 gram) three times a day, made up in capsule, cachet, or emulsion. In lieu of this, the pill containing 1 grain (0.06 gram) each of **squills, digitalis, and blue pill** may be tried. Free catharsis has been recommended, and may be secured by compound **jalap powder,** grains 15-60 (1-4 grams), by **magnesium sulphate,** or some of the **laxative saline waters.**

SURGICAL TREATMENT.—The surgical treatment consists in **tapping** when required. It has been thought by some that early tapping has a restraining influence over the disease. This is certainly very doubtful. A **drainage tube** was left in the opening by Hale White, but he did not see any benefit from this line of treatment. A **fistulous opening** has been tried but the risk of causing peritonitis is rather grave. A **Southey's tube** has been inserted into the abdominal cavity, and the opening of the abdomen and rubbing of the peritoneal surfaces tried, with the object of inducing absorption of fluid. **Talma's operation** or some modification of it as practiced by Drummond or Morrison may be tried. By this procedure the omentum is attached to the connective tissue of the abdominal wall, and collateral drainage is established. In many cases this has given marked relief.

SPECIFIC THERAPY.—As the writer holds that the disease is caused by a streptococcic infection, the **vaccine treatment** should be administered.

Prognosis.—The progress of the disease is very slow, covering periods of from two to sixteen years, according to Nicholls. Emaciation and asthenia finally become marked, and the patient usually dies of some intercurrent affection, such as peritonitis, pneumonia, empyema, pleurisy, pericarditis, or nephritis. In true hyperplastic perihepatitis, death always eventually occurs. As the disease advances the need for tapping becomes more frequent. Restoration of function can only occur to a very limited degree in the intervals of quiescence between tappings.

Pathology.—MACROSCOPICAL FINDINGS.—The gross appearance of the liver is very characteristic, as the surface of the organ is covered with a firm, glistening, whitish, fibrous coating, of considerable thickness. It is described as resembling the icing on a cake, hence the name "sugar-iced liver." On the surface of this coating depressions and cracks are usually visible, caused by the contractions that have taken place in it. This coating can be stripped off, leaving the peritoneal covering of the liver in a fairly normal condition. The liver is more or less deformed by the pressure and shrinking of this abnormal covering. This process may turn the anterior margin of the liver over upon the convex surface. As a result of the contraction of this fibrous coat, the gall-bladder is frequently quite small and empty. The biliary ducts, however, are not involved, which accounts for the absence of jaundice in nearly all these cases. There is very rarely compression of the portal vessels, leading to the conclusion that the ascites is due to the inflammation of the serous surface of the liver and to peritonitis, and not to vascular obstruction. The liver is soft, vascular and fatty, with lobular atrophy, but this atrophy is not a true cirrhosis. There may be a slight amount of fibrosis extending into the surface of the liver for a short distance. If the patient dies in the early stage of the disease the liver is covered with a layer of white lymph that can be readily removed. The capsule of the spleen is thickened and is adherent to the adjoining parts, due to serous inflammation. The kidneys are frequently congested, and in cases with arteriosclerosis they are usually contracted and granular. The peritoneum is very liable to become fibrosed and undergo hyaline changes similar to the covering of the liver. The omentum is thickened and is rolled up into a mass or rope-like body, while the mesentery is thickened and contracted, fastening the intestines close to the spine. The pleuræ and pericardium often reveal somewhat similar thickenings, contractions, and adhesions. These changes have given rise to the name *polyorrhomenitis* for the disease.

MICROSCOPICAL FINDINGS.—The microscopical changes are also very interesting. The fibrous covering of the liver is found to be laid down in thin layers, between which there are usually some white and some mast cells. Between the layers nearest the liver there are found a considerable number of leukocytes. The whole layer undergoes hyaline degeneration, and hence Nicholls calls it hyaloserositis. There are no blood-vessels in this morbid fibrous covering, nor is there any fibrin. The surface of the liver shows changes also. The capillaries are dilated, making the tissue appear vascular. There is pigmentary and fatty degeneration in the liver cells. The true glissonian capsule of the liver may show some fibrosis, which penetrates a short way into the liver. The surface layer of the liver tends toward hyaline changes.

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ACUTE NON-SUPPURATIVE HEPATITIS

(*Acute Parenchymatous Hepatitis*)

Etiology, p. 79—Symptomatology, p. 79—Diagnosis, p. 80—Treatment, p. 80:—Prophylaxis, p. 80; General and curative treatment, p. 81; Management of convalescence, p. 82—Prognosis, p. 82—Pathology, p. 82.

Etiology.—PREDISPOSING CAUSES.—Cold may act as a predisposing cause to the entry of the infection into the liver, though this has but little bearing in the cases of hepatitis occurring along with pneumonia, typhoid fever, or sepsis. Residence in hot countries and the tropics has no other relationship to the disease than placing the person where the *Plasmodium malariæ,* and *Amœba dysenteriæ* are found. Errors in diet, especially abuses in alcohol, no doubt predispose to hepatic infections.

In addition to the diseases mentioned above parenchymatous inflammation of the liver is frequently found in connection with erysipelas, malaria, and amebic dysentery.

EXCITING CAUSE.—The exciting cause is some infecting organism, or some irritant toxin carried to it through the hepatic artery or the portal vein. Thus we meet with cases caused by infectious diseases, by injurious materials—as alcohol, phosphorus, and certain metallic poisons—and toxins generated in the intestinal canal.

The septic organisms find their way into the system in many ways: the malarial plasmodium through the agency of the anopheles mosquito, and the *Amœba dysenteriæ* by drinking water or consuming food that has been infected.

Symptomatology.—CLINICAL HISTORY.—*Mode of Onset.*—The onset of this disease varies with the nature of the infection, toxin, or irritant causing it, though this stage is prompt enough to come well within the meaning of an acute illness of a few days' duration. There is frequently a sensation of chilliness, and a feeling of depression.

Symptoms During Progress of Disease.—Vomiting is liable to come on early in the attack, and, due to the severity of the retching, the vomitus may contain blood, but otherwise is of the characteristic greenish color. There may be diarrhea and the stools are very foul-smelling. There may be jaundice, but this is generally slight in amount, producing but little color in the conjunctiva or skin.

The patient complains of nausea and pain, and there is usually a feeling of great thirst. There is headache, sleeplessness, restlessness, and depression. There is a feeling of revulsion to food, and a sensation of fullness in the region of the liver. Frequently there is pain in the shoulder.

PHYSICAL FINDINGS.—The patient is frequently observed to sit up in bed. The jaundice may be very slight and amount to only a subicteric tint, but in a few cases it may become intense, resembling icterus gravis. The facial expression is one of anxiety and depression, and is usually somewhat sallow. The breathing is often shallow, due to restraint in the movement of the diaphragm.

The thermometer reveals the presence of some *fever*. Cantlie holds that a temperature of 100° F. (37.8° C.) and over indicates the presence of an inflammatory process, as against a congested state of the liver in which the temperature does not go above 100° F. The enlarged liver and the diminished movements of the diaphragm can be seen by the *x*-rays.

(*a*) *Palpation.*—Palpation reveals a tenderness in the liver, and a protective rigidity of the abdominal muscles. The gland is also found to be enlarged.

(*b*) *Percussion.*—Percussion still further confirms the enlargement of the liver, which may extend upward considerably above the normal limit, and downward as far as the umbilicus. Due to the infective cause in many cases, and to prior malaria in some, the spleen is found to be enlarged.

LABORATORY FINDINGS.—If there is any jaundice the urine will contain bile-pigment. It is also of high color and specific gravity, with abundance of urates and uric acid. The toxemia may cause albuminuria. An examination of the blood may reveal the plasmodium of malaria, and the *Bacillus typhosus* may be found in the blood and urine. The amebæ may be found in the feces, the portal blood, and in the liver.

Diagnosis.—The characteristics of the disease as already given will usually suffice to make the diagnosis. There may be difficulty, however, in distinguishing hepatitis from some other conditions of the liver. In the case of suppurative hepatitis there is usually an irregular temperature with sweats and chills, and the presence of leukocytosis will prove helpful in making the diagnosis. In *amebic hepatitis* there is no relative increase in the polymorphonuclears. In *phosphorus-poisoning* there is acute hepatitis with jaundice, but the hemorrhagic vomits and stools should enable one to draw the distinction, with the history of having taken the poison. *Acute yellow atrophy* is characterized by deep jaundice, extreme nervousness and delirium, the rapid course of the disease, and the early reduction in the size of the liver. In *malarial hepatitis* there is the history of chills and fever, and at the time of these paroxysms the liver and spleen notably enlarge. The malarial organism can be found in the blood. The *acute form of cirrhosis* may so closely resemble acute hepatitis that time is required to make the distinction.

Treatment.—PROPHYLAXIS.—The prophylactic treatment consists in avoiding unnecessary exposure, taking proper care to avoid the ingestion of such foods, drugs, and drinks as are calculated to irritate the liver, also taking precautions against becoming infected by the organisms of typhoid fever, malaria, and tropical dysentery.

General Management.—The patient must be put to **bed.** A **warm bath** is useful. The **air and temperature of the room** should receive due consideration.

Diet.—The diet should be of a very light, simple nature; and, at first, the comfort of the patient is favored by permitting water or some effervescent water only. Raw meat juice, lemon, albumin, jelly, chicken broth, rice and barley water, and whey and junket may be tried. Alcohol must be avoided.

Climate.—If the patient lives in the tropics or in a malarial district, removal to a temperate or non-malarial locality may become obligatory.

Local Treatment.—Local treatment for the liver is often useful in the form of an **ice-bag, a hot fomentation, leeching, dry-cupping, or strapping the side.**

Treatment of Symptoms.—The treatment of symptoms will claim attention. The pain may demand an opiate in the form of a hypodermic injection of grain 1/6 (0.011 gram) **morphin.** The vomiting may be relieved by the use of **bicarbonate of soda, bismuth** in some form, or **carbonate of magnesia.** One of the most successful methods is encouraging the patient to drink **hot water** freely. This flushes the stomach, and, when retained, fills the blood-vessels, inducing action of the skin and kidneys.

Medicinal Treatment.—Drugs are at times very useful. Foremost are the saline aperients in the form of some mineral water such as **hunyadi jános, Carlsbad,** or **Apenta. Magnesium sulphate,** drams 2-4 (8-16 grams) or **sodium sulphate,** in similar dosage, as required. **Calomel** and energetic purgatives should be avoided in the early stage of the attack. When vomiting and pain have subsided, repeated small doses of **calomel,** grain 1/4 (0.016 gram), **followed by a saline,** fill a useful place. If there is a history of malaria, **quinin** should be given as soon as the urgent stomach symptoms have abated. The dose should be a liberal one of 5, 10, or 15 grains (0.325, 0.650, or 0.972 gram), about one hour before the expected chill. This should be followed by moderate doses of a few grains thrice daily. Under this treatment, the liver may speedily return to its normal size. In amebic hepatitis 20-40 grains (1.30-2.60 grams) of **ipecacuanha** should be given twice a day, preceded by an opiate, to prevent vomiting. A suitable preparation of **opium** is 10 minims (0.6 c.c.) of the deodorized tincture. Musser and Kelly recommend **emetin hydrochlorid** in amebic hepatitis, in doses of from 1/3 to 1 grain (0.021 to 0.065 gram) administered hypodermically. **Chlorid of ammonium** in 20 grain (1.30 grams) doses is recommended by some in the acute stage. It cannot be given on a sick stomach. It may be combined with 5-10 minims (0.3-0.6 c.c.) of dilute **hydrochloric acid,** and 2 or 3 minims (0.12 or 0.18 c.c.) of **liquor strychninæ.** The recovery of cases complicated by syphilis is greatly aided by the administration of **iodids and mercurials.**

Surgical Treatment.—Surgical treatment by **withdrawing fifteen ounces of blood** from the liver by an aspirator has been advised. It is difficult to strike a vein of sufficient size, and it may become necessary to insert the needle several times before a successful result can be obtained. There is also the risk of hemorrhage into the peritoneal cavity. **Venesection** from the arm and the replacement of an equivalent amount of normal saline is much more simple and safe, and yields good results in suitable cases of the sthenic and plethoric type. **Free purgation** with saturated solution of **magnesium sulphate,** and the administration of **tincture of veratrum viride,** 15 minims (0.92 c.c.), every four hours for several doses, to empty the blood back into the portal vein, and the application of four or six **leeches** over the liver will meet all the indications that abstraction of blood by the needle is intended to achieve.

MANAGEMENT OF CONVALESCENCE.—The care of the patient during convalescence should receive attention. A light, nutritious, and non-stimulating diet must be recommended. Change to a suitable resort is advisable. A lengthy period of rest should be enjoined, and the person should live a strictly temperate life. All irregularities of the digestive organs must be corrected as far as possible, with the object of preventing recurrence of the disease by intestinal intoxication of the liver.

Prognosis.—This depends to a great extent upon the cause, whether a first or a subsequent attack, and the climatic conditions. Each attack renders the person more prone to the disease. If the person lives in the tropics the prognosis is much better if he can move to a temperate climate. Each attack disposes toward hypertrophy of the liver, and, finally, to cirrhosis. In uncomplicated attacks the outlook as to recovery is good; but in subsequent attacks the probability that damage to the liver will become permanent is considerable, and makes a guarded prognosis necessary. The mortality in first attacks is low, but in recurrent attacks it seriously rises with the occurrence of cirrhotic and atrophic changes in the liver. The function of the organ may be completely restored. In malarial and amebic cases the prospects in this regard are not so good, as in the former there is a tendency toward the formation of atrophic foci, while in the latter the risk of recurrence is considerable. In cases of severe acute hepatitis there is much danger to both life and function, as they approach icterus gravis in character. In tropical cases suppuration may occur.

Pathology.—The morbid changes found in the liver are very varied, according to the severity of the inflammation, and range all the way from what is found in active congestion to the destruction of tissue, as in acute yellow atrophy.

MACROSCOPICAL FINDINGS.—Observation shows the liver is enlarged in most cases, swollen and softened, and that it is paler than normal. On section it is also of a pale and opaque appearance. On the surface, in malarial cases, are seen many small nodules, and the gland, as a whole, may be pigmented from a brownish to a slaty color. When the liver is swollen and enlarged the edges are thickened and rounded. In some cases of a very acute nature and much infection, the liver is so softened that it appears to fluctuate. In some pyemic cases the lobules be marked out as bile-stained areas surrounded by grayish or whitish rings. A nodular condition of the liver has been sometimes found when there was no history of malaria. There may be collections of leukocytes along the portal sheaths, and the parenchymatous cells may become fatty, amyloid, and vacuolated.

MICROSCOPICAL FINDINGS.—The microscopic findings, like the macroscopic, vary greatly. The cells are cloudy and many of them are granular, consisting of albuminous and fatty matter, and the nuclei are frequently indistinct. There is sometimes marked pigmentation of the liver cells. There is, at times, an infiltration of the liver with leukocytes, plasma-cells, and young connective tissue, which is found to occur mainly around the portal vessels, in the foci of necrosis, and in more inflamed nodules. The small arteries, veins and capillaries are dilated, with a swelling and desquamation of their endothelium, and they often contain minute thrombi. There is a similar swelling, edema, proliferation and desquamation of the cells of the biliary ducts. The lymphatics share in these inflammatory changes. In cases that last for considerable time or are caused by a milder irritant, or are of the recurrent type, there may be considerable proliferation of the connective tissue, ending in cirrhosis. In cases of ameba, malaria, typhoid fever and sepsis the several organisms may be found in the liver. In these infections there are usually morbid changes in the spleen and kidneys.

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ACUTE SUPPURATIVE HEPATITIS

(*Hepatic Abscess, Suppurative Pylephlebitis, Suppurative Hepatic Phlebitis, Suppurative Cholangitis, Hepatic Pyemia*)

Etiology, p. 84—Symptomatology, p. 84—Diagnosis, p. 86—Complications, p. 86—Clinical varieties, p. 86—Treatment, p. 87:—Prophylaxis, 87—Prognosis, p. 88—Pathology, p. 88—Historical summary, p. 89.

Etiology.—PREDISPOSING CAUSES.—The predisposing causes are numerous. The residence in tropical climates has an important bearing on the production of the solitary abscess of the liver. Malaria may also lower the resistance of the gland. Errors in diet and the abuse of alcoholic beverages injure the digestive canal and also throw toxic and irritant materials into the liver. Diseases in other organs, such as gastric and intestinal ulceration, appendicitis, ulcerative endocarditis, septic wounds, empyema, abscess in the ear, Malta, yellow and typhoid fevers predispose to this condition, as do also sex—males suffering more frequently than females—diseases in the gall-bladder—as gall-stones, infective cholangitis—and age, the disease being one mainly of adult life.

EXCITING CAUSE.—The exciting cause is invariably the entry into the liver of pathogenic organisms. These may gain access by a punctured wound, or by being carried to the liver from some adjoining infective inflammation, or by the portal vein, the hepatic artery, the hepatic vein, the lymphatics, or the biliary ducts. The causative organisms are the varieties of the staphylococci and streptococci, the common colon bacillus, the *Amœba dysenteriæ* or *Entamœba dysenteriæ*, the typhoid fever bacillus, *Bacillus dysenteriæ*, *Bacillus pyogenes fœtidus*, *Diplococcus pneumoniæ*, actinomyces, and *Bacillus pyocyaneus*. It should be remembered that mixed infection may exist in a given case.

Symptomatology.—CLINICAL HISTORY.—The period of onset and the symptoms of the incubation or early period of the disease vary greatly. Attempts have been made to divide suppuration in the liver into acute, subacute, and chronic forms; but all such divisions must prove of little value, as a chronic case may suddenly take on acute manifestations and an acute form may become chronic.

Mode of Onset.—This varies greatly. When the abscess is caused by an injury or by extension from an adjacent part, the attack usually comes on abruptly. There is enlargement of the liver, which is irregular in shape. The gland is tender on pressure, and there are chills, sweats and fever, and there may be such pressure on the biliary ducts as may cause jaundice. Subjectively the patient feels pain in the region of the liver, often referred to the right shoulder, and increased by motion.

In pyemic abscesses of the liver the onset is generally sudden. There may be obvious changes in the size and shape of the organ, though these are not often at all marked, as the death of the patient may prevent much change in these respects. The chills, sweats and fever do not aid much in the diagnosis, as they are features of the infection from which the liver trouble arises. There may be some degree of jaundice. The chief subjective symptom is the pain.

Tropical liver abscess and those due to causes in the intestines are usually slow in their onset, and may be very obscure in their manifestations, though the history will enable one to form a correct opinion. The usual symptoms of tenderness and enlargement can be detected. The patient may complain of chilliness, pain and malaise.

Physical Findings.—The sufferer appears to be very ill, even though local symptoms are not pronounced. There is distinct nervous and mental depression. The color of the skin is a grayish-yellow, and the eyes may show an icteroid tint, but jaundice is only present in about 16 per cent. of the cases, caused by pressure in the biliary tubes. The patient lies in the dorsal position.

Palpation.—Palpation will reveal a swollen condition of the liver, and the outline is often irregular and bulging at one or more points. Tenderness will be elicited. The pulse is felt to be rapid and feeble, but, at the times of febrile paroxysms, may become tense. There may be extreme rigidity of the right rectus muscle, though this is usually only moderate and may be absent.

Percussion.—Percussion carefully conducted will outline the area of the dullness and its contour. It may also detect other foci of disease. Ascites, sometimes present, may also be discovered in this way.

Auscultation.—Auscultation may detect a friction rub when the peritoneum over the liver becomes inflamed. Heart lesions may also be noted, and these bear an important etiological relationship, especially ulcerative endocarditis.

Succussion.—This is a test of much value. If the patient is shaken, as in pyopneumothorax, pain is felt in the liver, and radiates to the right shoulder and across the upper zone of the abdomen.

Laboratory Findings.—The blood usually shows a leukocytosis, though this may be low or suddenly diminish, owing to a lowered resistance. Various bacteria have been found. The urine may contain albumin, and occasionally bile coloring. The pus from the abscess has been found to contain staphylococci, streptococci, pneumococci, *Bacillus typhosus*, colon bacillus, *Amœbæ dysenteriæ*, *Actinomyces bovis*.

Special Examinations.—The x-rays may be used to determine the size, shape, and movements of the liver. The aspirator may be employed, but the utmost care must be taken to prevent the spread of infection.

Diagnosis.—The diagnosis of suppuration in the liver is often a difficult matter. Special attention must be given to the *causative factors* and the *source of infection.* Pain in the liver, sometimes referred to the shoulder, enlargement and tenderness of the liver, occasionally jaundice, and the almost constant symptoms of fever, chills, sweats, and leukocytosis will establish the diagnosis. The symptoms in tropical abscess may be slight and latent, and the resistance may be poor and a low leukocyte count may follow.

Care must be taken to distinguish abscess of the liver from gallstones and inflammation of the gall-bladder, abscess in the adjoining wall, subphrenic abscess, empyema, gummata, suppurating hydatids, pyelitis of the right kidney, and enlargement of the liver associated with pernicious anemia, leukocythemia, ulcerative endocarditis, or scurvy. Soft, cancerous nodules might be mistaken for abscess, but there is less fever, and the history of malignant disease in some other part of the body would help to remove doubt. An echinococcus cyst develops slowly and without fever and the cyst is hard, tense and elastic, yielding a thrill on percussion.

If *aspiration* is resorted to as a means of clearing up the diagnosis, a fine needle should be used, and the skin and the instrument rendered sterile. When the needle has entered the soft parts the cock must be opened so that the pus can escape through the needle and not along its side, as it might do under pressure. The aspiration should be continuous. The small opening is sealed with some suitable dressing. If the puncture is carefully made, rarely will any harm result. The pus will yield valuable bacteriological information.

Complications.—The spleen, in this condition, is enlarged and may also become infected and be the seat of suppuration. The suppuration may extend into the lung, the pleura, and the pericardium. The liver abscess may rupture into the peritoneal cavity, or adjoining hollow viscera. There are cases on record where the pus was discharged into the kidney. Cerebral abscesses have complicated some cases. Strangulation of the small intestines has occurred around the adhesions, and adhesions have been so located as to give rise to jaundice by obstruction of the bile-ducts.

Clinical Varieties.—Two clinical varieties have been described, but they are liable to blend into each other in type.

The first variety is the single large abscess form, or the *tropical suppurative hepatitis.* Here, however, it must be noted that in this variety there may be more than one large abscess, or there may be a number of small ones forming around the primary large abscess, by infection.

In the second variety, the *non-tropical suppurative hepatitis,* or that caused by septic infection, there are usually many abscesses ranging in size from a pin's head upward. This variety may resemble the large abscess type, by a number of the small and adjacent abscesses coalescing into one.

The division of the disease into these two main varieties has some clinical value.

Treatment.—PROPHYLAXIS.—Much can be accomplished in the way of prevention. Persons who are living in a tropical climate must regulate their diet with care, and must avoid all abuse of alcoholic stimulants. If they have suffered from malaria and dysentery with hepatic complications, they should move for a time to a temperate region. Those who have any foci of active infection should have these thoroughly treated, and, perhaps, by this means avoid the formation of multiple pyemic abscesses in the liver.

Among the sources from which the liver may become infected one may mention septic wounds, infected gall-bladder, dysentery, appendicitis, gastric and duodenal ulcers, typhoid fever ulcers, ulceration of new growths in the intestinal canal, infected hemorrhoids, and suppuration in the pelvis. Infective endocarditis, gangrene of the lung, suppurative otitis, osteomyelitis, and fetid bronchitis may infect the liver through the general circulation by way of the hepatic artery. All these foci must receive due consideration.

GENERAL TREATMENT.—This must be of a supporting and restorative character. The patient should be given the benefits of a comfortable bed, a well-lighted and well-ventilated room, and nourishing liquid or light diet.

Diet.—The diet may be prepared in a variety of ways to render it as attractive as possible, and predigestion of the milk, gruels, etc., may be tried.

Climatic Treatment.—Those living in a malarial district or in the tropics, who have also been infected with malaria or who have suffered from amebic dysentery, should be well advised to seek a change of climate. This would be both prophylactic and therapeutic with regard to liver abscess. Large doses of **quinin**, grains 5-10 (0.3-0.6 gram), and of **ipecacuanha**, grains 20-40 daily (1.3-2.6 grams) might prevent the occurrence of abscesses in persons infected with these diseases.

Treatment of Symptoms.—This consists in measures for the relief of pain, the reduction of fever, the inducing of sleep, the regulation of the bowels, and the stimulation of the appetite.

Pain is best relieved by an opiate in some form, as grain 1/6-1/4 **morphin** (0.010-0.016 gram), or a good **tincture of opium**, minims 5-10 (0.3-0.6 c.c.). The *temperature* is best controlled by proper **sponging** with tepid, cold, or some evaporating affusion. The *bowels* should be moved as required by **enemata or mild aperients**. The *appetite* should be assisted by prescribing some simple bitter, such as **gentian, nux vomica, and calisaya**, or a combination of these, along with such a mineral acid as **dilute hydrochloric acid**, minims 5-10 (0.3-0.6 c.c.) three or four times daily. If the *hepatitis* is due to the ameba, **emetin hydrochlorid**, 1 grain (0.06 gram), should be given daily hypodermically for six to twelve days. It may also be given intravenously in severe cases.

SURGICAL TREATMENT.—As soon as an abscess has been diagnosed the indication exists for surgical treatment. Some writers, as Manson, Cantlie, Rogers, and Waring, have recommended aspiration. There are serious objections to this method, such as striking the portal vein, the vena cava, a hollow viscus, rupturing the abscess sac and permitting the escape of the pus into the abdomen, or causing free hemorrhage. Further, it does not secure perfect drainage and emptying of the abscess. The best ideals of surgery will be met by cutting down upon the liver, and exposing the diseased part to view, opening and draining the cavity.

SPECIFIC THERAPY.—For the multiple septic or pyemic abscesses surgical treatment is not suited. In such cases an autogenous vaccine might be tried.

Prognosis.—The prognosis is very unfavorable in the multiple abscess variety, as the mortality is very high, the duration of the illness being one to three weeks. In the large abscess variety the prospects for a recovery are much better, and Cantlie claims that, by proper aspiration, the mortality has been reduced from 80 per cent. to 20 or 30 per cent. The open surgical method with free drainage should still further reduce the death rate. Rupture of the abscess into the peritoneal cavity demands immediate operation, but rupture into the lung or into a hollow viscus may permit of some delay while watching the progress of the case closely.

Pathology.—MACROSCOPICAL FINDINGS.—The macroscopical appearances will differ according to whether the case is one of multiple pyemic abscesses or of one or two large abscesses, as in the tropical disease. The appearance will also vary with the stage of the disease, and the location of the principal foci, and the nature of the organism.

In the small pyemic abscess variety, the result of infection carried by the portal vein, the hepatic artery, emboli in the hepatic vein, or extension from suppurative cholangitis, the liver is enlarged, swollen, and presents the appearance of a general inflammation. There will be seen numerous grayish or yellowish spots, surrounded by a congested zone. The softened spots vary greatly in size from 1 millimeter to 10 centimeters. Many of these grayish softened spots discharge pus on section, leaving cavities to view, with irregular walls. The contents of these cavities may be thickish or quite thin, blood-stained, or greenish from bile. The pus may be very offensive, and the vessels may be the seat of suppuration. In the amebic variety the contents of the abscess are usually viscid.

In the formation of a single abscess there is a pale, softened area, which may be irregular in shape. This area in time softens into an abscess. Immediately around this area there is a margin of softening substance of a buff color, usually intensely hyperemic, undergoing degeneration or necrosis. This process extends outward from the abscess and continuously destroys more liver tissue. In older abscesses there may be a lining in the abscess walls of granulation tissue of a very irregular and ragged appearance; outside this layer there may be considerable fibrous tissue. The rest of the organ is enlarged. When there are only one or perhaps two abscesses, the contour of the liver may be quite uneven. Shreds of liver tissue may be found as part of the contents of these abscesses. Owing to the adhesions that are liable to form, the liver may be bound to adjoining structures. The size and shape of the abscesses may vary much. As much as 19 pints of pus have been present. If two or more abscesses coalesce the shape may be quite irregular. After the evacuation of the pus the liver is still heavier than in health. Occasionally the liver is yellow in color, resembling the condition found in phosphorus-poisoning.

MICROSCOPICAL FINDINGS.—The microscopical appearances also vary with the variety and the stage of the disease. At an early period in the multiple abscess variety the capillaries are markedly dilated, there is leukocyte infiltration, vascular thrombosis, and, perhaps, coagulation necrosis. The infecting bacteria may be found. The leukocyte infiltration is influenced by the channel carrying the infection. If it is borne by the portal circulation it will be noticed first in the interlobular tissue, and, later, along the intralobular branches of the portal vein. In infection by the arterial route the central parts of the lobules show the leukocyte infiltration first, and the interlobular tissues later. If the infection finds its way along the biliary tubes, the infiltration first appears around the small branches of this system. As the disease advances these distinctions may wholly disappear in the general infiltration that ensues. The parenchyma also shows important changes. The cells swell and become cloudy; they often contain an excess of coloring matter. The nuclei do not stain readily or not at all and they begin to break up—conditions to which the terms karyolysis, plasmolysis, and karyorrhexis have been applied. In time there is nothing but a liquid mass, containing leukocytes, liver cells, shreds of tissue, coloring matter, fat globules, bacteria, etc.; all this is surrounded by a pyogenic wall already described.

In the amebic or solitary abscess variety the microscope shows that at first the capillaries are dilated, filled with blood, and may contain the parasite. When pus has been formed it is surrounded by a necrotic liver area in which are found a few leukocytes, red blood-corpuscles, fibrin filaments, and amebæ. Beyond this necrosing area there is a margin of greater connective formation and, in old abscesses, this may become quite firm. In the hyperemic area surrounding these abscesses are frequent small hemorrhages. The pus is notable for the low white count, and that it is made up mainly of liver cells, red blood-corpuscles, and amebæ. Pus-producing bacteria are absent in about 80 per cent. of the cases.

Historical Summary.—Morgagni, about 1750, noted the occurrence of abscesses of the liver, and made the suggestion that they might be caused through portal circulation. Cruveilhier, in 1852, described abscesses of the liver following operations for hernia and hemorrhoids.

Budd, Cheyne, Annesley, and Abercrombie, in the first half of the nineteenth century, connected abscesses of the liver with dysentery. The view of Budd gradually came to be accepted, that dysentery was the cause of many cases of hepatic abscess. The excellent work of Shiga, in Japan, and Flexner, in the Philippines, shows that most cases of dysentery are caused by a bacillus of the typhoid fever group, though differing from the one producing the fever of that name. These are acute cases and are seldom complicated by abscesses. When this occurs the dysentery is of the chronic type and due to the ameba. On account of the variety of the organisms causing liver abscesses the disease is world-wide.

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CHRONIC INTERSTITIAL CIRRHOSIS

(*Laennec's Cirrhosis, Atrophic Cirrhosis, Alcoholic Cirrhosis, Granular Cirrhotic Atrophy, Gin- or Whiskey-drinker's Liver, Multilobular, Annular, or Venous Cirrhosis, Hobnail Liver, Portal Cirrhosis*)

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Etiology.—Predisposing Causes.—*Age.*—Chronic interstitial cirrhosis is a disease of adult life. While it may occur at any age from childhood to old age, yet the vast majority falls into the period of thirty to sixty years, the age at death being on an average forty-seven to forty-eight years.

Sex.—Of the cases, 77.4 per cent. are males and 26.6 per cent. are females.

Occupation.—Occupational Diseases.—Occupation, apart from the liquor traffic, plays a minor part in its etiology; but it has been claimed by some authors that chronic lead, silver and arsenic poisoning have given rise to the disease. The absorption into the portal circulation of minute carbon particles, which in this way are carried to the liver, has appeared to be the cause of some cases.

Spices.—Highly spiced foods and drinks seem to be the cause of this form of cirrhosis in such countries as Egypt and India.

Toxins.—Cases also appear to arise from poisons generated in the intestinal canal, such as the fatty acids and food that is undergoing decomposition.

Alcohol.—Alcoholic beverages are the cause of many cases of cirrhosis. It has now, however, been definitely proven that ethyl alcohol plays a very small part in this causation, and that the pathogenic influence of these beverages is due to other constituents which they may contain. Alcohol in itself can only give rise to a fatty degeneration of the liver. But the abuse of alcoholic drinks is indirectly potent for evil by inducing intestinal irritation, from which arise many toxic and irritant bodies.

red corpuscles may fall to 1,300,000, as recorded by Cabot, and the hemoglobin be only 22 per cent. of the normal.

The ascitic fluid is alkaline and is clear, pale yellow, slightly reddish from bile coloring, or greenish in color. Its specific gravity varies from 1.007 to 1.016, according to the observations of the writer. It contains about 0.5 per cent. albuminous material, and there may be a trace of urea, urobilin, or sugar. There is a large amount of the chlorids in it. In this ascitic fluid endothelial cells predominate, but if the patient has been tapped there may be fewer endothelial cells and more lymphocytes. A marked increase in the latter should raise the suspicion of tuberculous peritonitis. When peritonitis is present the fluid is liable to become flocculent and contain cloudy matter. The ascitic fluid may be chyliform or fatty, or milky and non-fatty. It may also contain blood, the tubercle bacillus, or the Bacillus coli communis.

SPECIAL EXAMINATIONS.—The functional activity of the liver has been tested by giving the patient a sugar meal. It is known that the healthy adult, when fasting, can take 100 grams of sugar without showing alimentary glycosuria. It was thought that the cirrhotic liver might have lost some of its glycogenic function, and such a quantity of sugar as that mentioned would reveal functional failure. Such, however, has not been found to be the case unless there is accompanying disease of the pancreas. Alimentary levulosuria is a test of considerable value, however, as shown by a number of observers, some claiming that it is almost constant in cirrhosis of the liver.

X-rays and screening may be employed to determine the size, location, shape, and movements of the liver, and the size of the spleen.

Diagnosis.—The clinical history may be all that one has for guidance in the early period of the disease. The abuse of alcohol, especially the ardent spirits, and particularly if drunk on an empty stomach, should be made the subject of inquiry. Wine and malt drinks are capable of causing the disease. The morning nausea and vomiting, of the drinker's type, the dyspepsia, the pain and uneasy feeling in the region of the liver, the occurrence of a slight degree of fever, the presence of an icteroid tint in the skin, and urobilinuria should enable one to arrive at a fairly satisfactory diagnosis. When hematemesis, ascites, melena, and the characteristic expression have appeared there can be no doubt. Age is an important factor, as the average at death is about forty-seven years, though it has been met with in both the young and the old, a few children being its victims. One-third of the cases occur among females.

Hematemesis in cirrhosis of the liver must be differentiated from that occurring in gastric and duodenal ulcers, and in cancer of the stomach. The hyperchlorhydria of the gastric ulcer, the relationship of pain to the taking of food, the relief obtained by vomiting, the absence of enlargement or tenderness of the liver, and the age and sex of the patient will help to clear the diagnosis. In the case of carcinoma ventriculi, the hematemesis is usually frequent and small in amount; there is presence of lactic acid and absence of free hydrochloric acid; a tumor in the stomach may be felt; and there may be evidence of pyloric obstruction.

The ascites of hepatic cirrhosis, present in 80 per cent. of the cases, must be distinguished from that caused by cardiac disease, tumors in the abdomen, chronic inflammation of the peritoneum however caused, perihepatitis, kidney disease, and cachectic conditions. Examples of portal obstruction by a thrombus give rise to rapidly forming ascites. The causes of ascites, apart from the liver, are diseases of the peritoneum, obstruction of the portal vein, tumors and enlargements of adjacent organs, obstruction of the inferior vena cava, valvular disease of the heart, kidney disease, and the essential anemias. The diseases of the liver that may be accompanied by ascites are atrophic cirrhosis, hypertrophic cirrhosis, syphilis, perihepatitis, hydatids, malignant growths, and hyperemia.

PHYSICAL FINDINGS.—The physical examination must be made with great thoroughness. When syphilis is suspected, the history of the case, the presence of some of the stigmata of the disease, and the Wassermann test should remove doubt. If there is any enlargement in the liver it is almost certain to be quite irregular. The closest attention must be given to the spleen. If it is found to be enlarged in connection with gastric disturbance, and no other disease, such as malaria, Banti's, or leukemia, it must be accepted as very strong evidence in favor of hepatic cirrhosis. Later on when there are hematemesis and ascites, the enlargement of the spleen is still more conclusive. The lungs, the testicles and other organs should be thoroughly examined with the object of determining whether or not there is tuberculosis. Should such be found it might throw light upon the cause of the ascites. The abdomen must be carefully palpated and percussed in search of new formations such as carcinoma, papilloma of the ovary, fibroid tumor of the uterus, as the possible cause of the ascites and other conditions present. The heart must be auscultated, as disease in it may cause hyperemia of the liver and the accumulation of fluid. Pylephlebitis and thrombus of the portal vein are much more acute causes of ascites than cirrhosis of the liver, while chronic universal perihepatitis is much slower. Cancer of the liver is more rapid than cirrhosis; there is more pain; a greater tendency to jaundice; the enlargement is irregular and is frequently considerably greater than that of cirrhosis, while the spleen is rarely increased in size. The possibility of amyloid, tuberculous, or other chronic disease of the kidneys must be remembered.

LABORATORY FINDINGS.—The blood should be carefully tested when there is enlargement of the spleen, with the object of excluding the essential types of anemia. The urine must also be examined for abnormal constituents, such as urobilin, tyrosin, leucin, indican, and levulose. The latter is of special interest as revealing loss of hepatic function. The ascitic fluid may yield valuable information. In hepatic cirrhosis the specific gravity seldom rises above 1.015, and contains from 1 to 3 per cent. of albumin, and most of the cells are endothelial. In the case of inflammatory exudates, the fluid is more or less turbid, and may be purulent or bloody; the specific gravity is liable to be 1.015 or higher; the albumin will range from 3 to 6 per cent.; there will be a predominance of lymphocytes and leukocytes. When the lymphocytes are very numerous—about 90 per cent.—the probability is tuberculosis, and when the polymorphonuclear leukocytes are abundant some pyococcic infection is present.

SPECIAL EXAMINATION.—As a further means of clearing away all doubt, where ascites exists with obscure conditions, the abdomen may be opened, the fluid drained off, and the viscera explored, as suggested by H. P. Hawkins. The opening may be used for the performance of the Talma-Morrison operation, if it be deemed wise to carry out such a procedure.

DIFFERENTIAL DIAGNOSIS.—*Biliary Hypertrophic Cirrhosis.*—In this disease the enlargement of the liver and spleen is greater than in portal cirrhosis, and the jaundice is always present, is pronounced in degree and continuous, whereas in the portal type the jaundice occurs only once in three cases, is mild in form and frequently disappears.

Malarial Enlargement.—In cases of prolonged malarial infection the spleen and the liver become enlarged, and the latter is tender to pressure. There is rarely true jaundice, there is marked debility, there is the history of chills and fever, and the plasmodium may be found in the blood. The results of quinin treatments confirm the diagnosis.

Malignant Tumor.—It is very difficult to distinguish between cancer of the liver with jaundice and ascites, and the late stage of cirrhosis with these two conditions. Enlarged glands may be discovered, and there may be a primary focus. A history of alcoholism, dyspepsia, and hematemesis, with enlarged spleen, point to cirrhosis. If the abdomen is opened, palpation will remove doubt.

Syphilitic Liver.—The history of the case and a positive Wassermann should enable one to make a diagnosis. In syphilis the enlarged liver is painful and tender, and its shape is usually irregular, but in cirrhosis both main lobes are affected. A course of treatment diminishes the size of the syphilitic liver.

Splenic Anemia.—In this condition the splenic enlargement and anemia are more pronounced than in cirrhosis, and there is a leukopenia. In splenic anemia there may be hematemesis, and the disease may end with hepatic cirrhosis. The distinction must rest mainly on the large spleen and the severer anemia of the splenic disease.

Kala-azar.—This form of enlarged liver can be distinguished from others by the presence of the Leishman-Donovan body.

Enteric Fever.—The enlarged liver, the fever and diarrhea of enteric fever may resemble very closely the enlarged liver, diarrhea, and fever that may follow a drinking bout in one who has a cirrhotic liver. Time will clear up the case.

Hepatic Abscess.—The history of living in the tropics or suffering from dysentery, or some septic condition, the irregular shape of the liver, and its marked tenderness, together with a leukocytosis, are the chief points of difference.

Hydatid Liver.—The enlargement is localized and uneven. The patient is in good health, and there is no hematemesis or jaundice. The hydatid liver is more prominent than the cirrhotic one.

Complications.—The complications that occur in hepatic cirrhosis are to a great extent the result of the causes of the disease. The alcoholic habit undermines the health and paves the way for the invasion of the *Bacillus tuberculosis,* which may be manifested in disease of the lungs, pleuræ, or peritoneum. From the abuse of alcohol may arise kidney disease, arterial degeneration, neuritis, mental derangement, delirium tremens, chronic dyspepsia and gastritis. Thrombosis of the portal vein is a rare complication. The reduced resistance favors the occurrence of infection, and the end of the patient is not infrequently due to some form of terminal infection. At any time during the progress of the disease the patient may develop a condition closely resembling uremia, for which it has been mistaken. The nature of the poison is not known. This condition may come on when the patient is not jaundiced and when he has not been drinking for weeks. Troublesome bronchitis may also coëxist. In addition to the hemorrhages from the nose, stomach and bowels, there may be hemoptysis and menorrhagia.

Association with Other Diseases.—Other diseases may have existed when the cirrhosis makes its appearance, or they may come independently of the condition of the liver. Prior to the onset of the hepatic disease there may have been a cancerous growth in one of the abdominal viscera; or such may commence subsequently to the liver disease. So it may be with gastric or duodenal ulcer. In like manner heart disease may precede or follow the onset of the cirrhosis. Bright's disease may precede the disease in the liver, or they may both result from a common cause.

Clinical Varieties.—Some authors have been disposed to subdivide true portal hepatic cirrhosis into several varieties, more, however, from the standpoint of etiology than of any essential difference in the clinical course or pathology. Thus we learn of the *alcoholic* and *non-alcoholic type,* and the view is put forth that the alcoholic variety is essentially an enlarged liver, while in the non-alcoholic variety the liver is smaller than normal. Sometimes in the diabetic with a cirrhotic liver there is a marked degree of pigmentation which has given rise to the name *bronzed diabetes* or *pigmentary cirrhosis.* In these cases there is a deposit of coloring matter in the liver, pancreas, secreting glands, lymph-glands, intestines and skin. The pigment in the internal organs contains iron, while the one deposited in the skin is iron-free. A *malarial variety* has been described. In severe cases of malaria there may be extensive destruction of the biliary cells, and a resulting fibrosis of the liver. These cases are accompanied by distinct pigmentation of the liver cells. It is very doubtful if malaria ever causes true cirrhosis of the liver, but it may give rise to the foregoing changes in cases where cirrhosis already exists. *Cirrhosis in children* has been designated as a variety possessing some differences from the disease in adults. Persons who swallow quantities of fine, irritating dust, as carbon, may develop cirrhosis. The liver may be black—*anthracotic cirrhosis.*

Treatment.—PROPHYLAXIS.—Prophylaxis is the ideal treatment. This, however, would cover a wide range of preventive therapeutics, and would include abstinence from alcoholic abuses, the adoption of a proper dietary, especially for the young, eliminating too highly spiced foods, the prevention of syphilis, more particularly as it affects the unborn, and a thorough regulation of the digestive functions to obviate auto-intoxication. But such a mode of living must, to a great extent, remain utopian. Though this is true, a careful regulation of diet and of the use of alcohol can accomplish much toward the prevention of the more severe and dangerous features of the disease. The **withdrawal of alcohol** will often arrest the progress of the disease.

GENERAL MANAGEMENT.—*Diet.*—The diet must be **light** and **simple.** It must be adequate for the nourishment of the body, but all excesses must be avoided. Starchy and saccharine foods should be reduced to the minimum, on account of the impaired condition of the liver. Meats, with the exception of some of the white sorts and some light, plain boiled fish, should be avoided, as well as rich soups. The non-starchy fruits and vegetables may be allowed. Milk, in some form, should constitute the main portion of the diet, and three to four pints for an adult is a fair allowance. The milk may be modified in many ways to meet the wishes of the patient. Thus it may be given as junket, buttermilk, with some effervescent or alkaline water, as a milk soup, or on vegetables and light puddings. Kumiss and skimmed milk may be found useful. A milk diet may be maintained for many months. As convalescence becomes established a greater variety may be allowed, such as egg, light farinaceous and protein food, but always of the simple and non-stimulating sort. Half a pint of water, half a pint of buttermilk, four pints of fresh milk, and one ounce of loaf sugar, kept in a warm place for thirty-six hours, has given satisfaction.

Alcohol.—Alcohol in every form must be forbidden, even in cases where the cirrhosis is not of alcoholic origin. It is unquestionably objectionable in the cirrhosis caused by other toxins as well as in the form due to alcoholic abuses. Even medicinal preparations should contain no alcohol, and drugs prepared without alcohol should be prescribed. To the foregoing statement a slight exception may be made in the case of patients in the late and adynamic phase of the disease. The use of alcoholic stimulants to such persons may help to prolong life and ameliorate some of the symptoms.

Local Treatment.—The application of a few **leeches** over the liver, or **dry cupping** may relieve the pain and sense of fullness. The employment of the **trocar** to withdraw blood from the liver or spleen is *not to be recommended,* as it is both dangerous and of very doubtful value. **Massage of the liver and abdomen** has been put forward as a means of reducing the portal congestion; but it is not trustworthy and must be *used with care.*

Treatment of Symptoms.—Dyspepsia is usually present and may give rise to much discomfort. The diet must receive attention, and may have to be varied to meet the conditions. If the dyspepsia is of the irritable type, **bismuth, bicarbonate of soda, and dilute hydrocyanic acid,** will be found valuable. In cases of *loss of tone,* dilute **nitrohydrochloric acid, strychnin, and other vegetable bitters, with pepsin,** should be tried, preferably after food. In case there is *flatulence* some of the intestinal sedatives and antiseptics should be ordered, such as **creosote,** minims ¼-5 (0.016-0.33 gram), **thymol,** grains 1-5 (0.065-0.325 gram), **bismuth salicylate,** grains 5-15 (0.325-1 gram), **salol,** grains 5-15 (0.325-1 gram), or **beta-naphthol,** grains 3-10 (0.195-0.65 gram). Small doses of **calomel,** grain 1/40-1/20 (0.0016-0.0032 gram), two or three times daily, may prove very serviceable.

Gastritis and the vomiting caused by it are best treated by **starvation** until the urgency subsides, aided by the administration of **subnitrate of bismuth, bicarbonate of soda, dilute hydrocyanic acid,** and light doses of **morphin,** grain ⅛ (0.008 gram). The application of an **ice-bag** or a **mustard leaf** has been found useful. When food is resumed it should be of a very bland character and of small amount, such as peptonized milk, albumin water, whey, and rice or barley water, cool to cold.

Constipation may call for attention, and is best relieved by gentle **exercise** when possible, light **massage** of the abdomen, and the drinking of **water freely.** When aperients become necessary, some mild saline should be selected, and **magnesium sulphate, sodium sulphate, sodium tartrate,** or one of the laxative mineral waters, meet the requirements better than the more drastic purgatives. Sometimes, however, the bowels are hard to move and these latter drugs may have to be employed. The following combination, in pill or capsule, meets the requirements in a very efficient manner:

Aloini	grain i (0.065 gram)
Rhamni purshianæ extracti (cascara sagrada)	grain i (0.065 gram)
Nucis vomicæ extracti	grain ss (0.0325 gram)
Belladonnæ extracti foliorum	grain ss (0.0325 gram)
Ferri sulphatis exsiccati	grain i (0.065 gram)

This dose may be repeated as the indications demand.

Diarrhea in the early stage may be the result of alcoholism. The drinking of alcohol must be forbidden, the diet must be strictly regulated, and some mild astringent ordered, as **bismuth subnitrate or carbonate,** grains 15 (1 gram), **pulverized aromatic chalk with opium,** grains 10-30 (0.650-1.95 grams), every four to six hours; **aromatic sul-**

phuric acid, minims 5-20 (0.3-1.25 c.c.), is worthy of trial, and **tannoform,** grains 5-15 (0.325-1 gram), is a useful non-irritant astringent. Diarrhea in the late and toxemic stage of the disease is useful and should not be checked. When it assumes a severe form it is usually fatal.

A *comatose condition from toxemia* is best treated by injecting into a vein or the muscles a pint or quart of **normal saline solution,** to be repeated in twelve or twenty-four hours if necessary. *If diacetic acid is present,* 2 drams (8 grams) of bicarbonate of soda to the pint should be employed. Copious saline or alkaline enemata may be tried. **Hot-air baths** may induce free perspiration, which may be materially increased by the hypodermic injection of **pilocarpin,** grain 1/8 (0.008 gram). **Caffein, camphor, or strychnin** should be combined with the pilocarpin. *Other nervous symptoms,* as mental disturbances and delirium, are best managed by **elimination through the skin, kidneys, and bowels.**

Hematemesis demands prompt and energetic treatment. The patient must be put to **bed** and **all food and liquids withheld for two or three days.** Ice in the mouth and sips of cold drinks are injurious, as they introduce liquid into the stomach and reduce the temperature. The *thirst,* which is usually very distressing, can be relieved by **water enemas** every four to six hours, or by pledgets of **cotton-wool wet with water** and placed in the mouth. **Normal saline solution** may be injected into the subcutaneous tissue. The injection beneath the skin of **morphin,** grain 1/6-1/4 (0.010-0.016 gram), is one of the most valuable remedies we possess, as it allays nervousness, quiets the circulation, and relieves any pain that may be present. **Digitalis, ergot, and adrenalin** are **injurious** as they raise the blood-pressure. Drugs that lower the blood-pressure, such as **amyl nitrite, sodium nitrite, erythrol tetranitrate,** are more likely to benefit the patient. **Calcium chlorid or lactate** may be added in dram (4 grams) quantities to one or more of the water enemas, or with the subcutaneous injections. The custom of ordering **lead, tannic acid, perchlorid of iron, turpentine, alum, nitrate of silver, and other astringents** by the mouth cannot be too **strongly condemned.** They cannot act in the stomach in such concentration as would contract a bleeding vessel, and they are very likely to induce nausea and vomiting. Five ounces of a 2 per cent. solution of **gelatin** may be injected deeply into the gluteal region. Great care must be taken that the solution is absolutely sterile. It is very doubtful if any preparation of the suprarenal gland can be given by mouth in such quantities as to act locally in the stomach as a hemostatic when one remembers the size and motions of the organ and the amount of gastric fluid, mucus and blood which it is certain to contain. It has been recommended and may be given in doses of grains 5 (0.3 gram) of the gland, or minims 30 (2 c.c.) of the 1 in 1,000 solution in a dram of water. The writer, many years ago, advised the placing of elastic bands around the extremities. These retarded the return of blood to the body, and lowered the blood-pressure in the viscera. These bands can be gradually removed. The lower bowel should be emptied by a **soap-and-water enema.** As soon as the stomach will permit of it, a laxative should be given to reduce the fullness of the portal vessels. The patient must be fed by the rectum. While recovery is taking place, the greatest care must be taken to restrict the diet to the blandest articles.

Melena is a frequent occurrence. In many cases this condition is due to blood that passes from the stomach into the intestines, and would call for the same line of treatment as that for hematemesis. When there is reason to believe that the bleeding is from some portion of the intestinal canal, the treatment is **rest,** the use of **opium** in some form to control peristalsis, and the administration of an astringent, as **pilulæ plumbi cum opio,** grains 2-4 (0.13-0.26 gram), **compound powder of kino,** grains 5-20 (0.32-1.3 grams), or **pulverized aromatic chalk with opium,** grains 10-30 (0.65-2 grams). A mixture containing **bismuth, tincture of opium, and chalk mixture** answers well. Food may have to be withheld for a time, and resumed with caution.

Epistaxis, hemoptysis, hematuria, and metrorrhagia may occur during the course of the disease. They are due to a congested state of the organs from which the blood escapes, to an impaired condition of the vessel walls, and to a lowered coagulability of the blood. A **brisk saline purgative** is indicated, also a restriction of the fluids taken.

For a low fibrinogen content in the blood a **milk diet** should be prescribed, and several doses of **calcium lactate,** grains 15-30 (1-2 grams), every two to four hours.

Ascites is sometimes removed by medicinal treatment, but frequently it fails. There are few drugs that can be relied upon to exert any very special action over this feature of liver cirrhosis. A **strictly milk diet** has been known to remove an extensive accumulation of fluid. For a time liquids other than milk should be taken in very limited quantities. The drugs that may be thought of are **spartein, adonidin, caffein, digitalis, theobromin, scoparin, Apocynum cannabinum, calomel, squills, Guy's pill** (1 grain or 0.06 gram each of squills, digitalis, extract of hyoscyamus and blue mass), the **citrate, nitrate, acetate and bitartrate of potassium,** solution of **ammonium acetate, elaterium, magnesium sulphate, and hydragogue cathartics.** The utmost care must be exercised not to reduce the patient's strength in the effort to reduce the ascites by these drugs. Perhaps the most successful general treatment of the ascites caused by hepatic cirrhosis is **rest in bed,** an **exclusive milk diet,** both as nourishment and drink, and such an amount of **magnesium sulphate** as will move the bowels three or four times daily. The writer prefers giving this drug in the dry form in large capsules. It is his experience that it may be administered for long periods without causing either irritation or exhaustion. When the ascites does not disappear in a reasonable time under the foregoing treatment, or shows a tendency to increase, recourse should be had to **tapping.**

The arguments that have been advanced against paracentesis abdominis carry but little weight to-day. These objections were the following: the risk of peritonitis, the removal of such a large quantity of albumin as the fluid contained, and the fact that the exudate soon formed again. With proper antiseptic precautions there is no likelihood of the first danger. As to the second objection, the albumin in the ascitic fluid is performing no useful purpose, and its removal is no added loss to the patient. The filling again of the peritoneal cavity can be averted to a considerable extent, or entirely, by proper treatment, such as **rest,** restricted **diet, suitable aperients and diuretics,** and carefully adjusted pressure around the abdomen by a **neatly fitting bandage.** The objections to leaving the fluid too long are very weighty, such as damage to the vitality of the peritoneum, the pressure effects upon the abdominal and thoracic viscera, and the disturbed action of the heart and the lessened flow of urine that results from this pressure. The indications for tapping are discomfort on the part of the patient, decrease in the amount of urine, edema of the legs, difficult breathing, the presence of râles in the lungs, and the onset of hemorrhages. In such a condition, the removal of the fluid will help greatly to relieve the engorged condition of the viscera, and ameliorate these features of the disease.

ORGANOTHERAPY.—Preparations of liver have been tried. Some have reported benefit, as shown by an increased output of urine and urea, and a reduction in the ascites and urobilin. **Hepatin** may be used hypodermically, or from 2 to 4 ounces of **pulped liver** taken daily.

MEDICINAL TREATMENT.—There are no specific drugs in this disease. **Ammonium chlorid** may relieve the congestion and pain of the early stage. **Bicarbonate of soda** is useful if the urine contains diacetic acid, and **mercurials and iodids** when syphilis is suspected or known to be present.

SURGICAL TREATMENT.—This consists in **tapping the abdomen,** or the performance of such an operation as will assist in establishing a free collateral circulation, and, thereby, control or cure the ascites. Tapping should be performed under strict antiseptic precautions. A small **trocar and cannula** should be employed, as it is desirable that the escape of the fluid should be slow. Two points are usually chosen. One is the midline half-way between the umbilicus and the pubis; the other is the midpoint of a line connecting the left anterior superior spine of the ilium and the umbilicus. The patient should be recumbent in the dorsal position with the head and shoulders gently elevated. The place selected for the paracentesis must be dull. The skin may be anesthetized with cocain or ethyl chlorid. The bladder must be empty. The cannula is fastened *in situ* by means of strapping, and a rubber tube attached to it through which the liquid flows to a vessel beside the bed. The abdomen should be compressed by a suitable binder, which is tightened as the fluid escapes. With the object of preventing the recurrence of the fluid, 1 dram (3.75 c.c.) of 1:1,000 adrenalin solution in 4 drams (15 c.c.) of water is thrown into the peritoneal cavity before the cannula is removed. The attempt to establish continuous drainage must be condemned.

Surgical operations for the purpose of establishing a free collateral circulation have been devised by Talma, Drummond and Morrison, Roberts, Paterson, and others. The operation is to make an incision parallel with the border of the ribs on the right side. The undersurface of the diaphragm is scraped and roughened, and the same is done with the convex surface of the liver. These surfaces are brought into contact by stitching the round ligament to the abdominal roll, and sometimes by stitches passing through the substance of the liver. The great omentum should be placed between the liver and the diaphragm as a means of still further increasing the vascular channels. Omentopexy or epiplopexy is another operation with a similar object in view: By it the great omentum is fastened to the abdominal wall, or it may be inserted between the peritoneum and the muscles. As a rule, and especially if there is much fluid in the abdomen, the patient should be tapped before this operation is performed. This operation ought to be performed before the disease has advanced too far, and the liver and general health have suffered too much damage. The diversion of a considerable portion of the venous blood from the liver enables the latter to detoxify more completely that which does pass through it, and the blood stream is improved. The liver is less congested and a larger share of arterial blood can enter it, increasing its nutrition and favoring cell regeneration. If the disease has reached an advanced stage, and is complicated by renal, or cardiac disease or jaundice, the operation is contra-indicated.

MANAGEMENT OF CONVALESCENCE.—The indications for the treatment of these patients during convalescence is summed up in strict **abstinence from alcohol,** a light, easily digested, and **nutritious diet,** careful directions as to the amount and sort of exercise, and the **avoidance of excitement** and nervous tension and worry. Care must be given to the action of the skin, the kidneys, and the bowels. Complicating disorders, if present, require attention.

Prognosis.—The prognosis as to ultimate recovery is very gloomy. Even in such cases as appear to make a satisfactory recovery the reserve power of the liver is seriously impaired. One of the best results the writer encountered was in a patient who lived for fifteen years after being confined to bed for several months with extensive ascites; but his health never became good. He had been a heavy drinker. From one to four years is the usual period, but acute cases in the young may reach a fatal termination in a few months, with symptoms resembling icterus gravis. The prognosis is influenced by the period of the disease when the patient comes under treatment, and the cause of the cirrhosis. The response to treatment is also important. Some cases react very poorly. Age is also a factor, as the outlook is very bad in children, the aged, and in acute cases in the young adult brought on by heavy drinking. The size of the liver should be noted, as those cases with considerable enlargement do much better than those with atrophy.

The mode of death varies. Sometimes the fatal ending is caused by hemorrhages from the stomach or bowels. There may be chronic renal disease associated with the cirrhosis of the liver. Some acute or terminal infection may carry off the patient. Most of the cases, however, become extremely weakened and emaciated and pass into a comatose state like that of uremia.

The prognosis as to function rests upon two main features: the increase of the normal anastomoses that exist between the portal and systemic veins, and the regeneration of hepatic cells. Judicious treatment that lessens intoxication of the liver, relieves congestion, promotes the health of the patient, and removes complications, aids both of these life-saving processes. The prognosis is now much more hopeful than it was some years ago.

Pathology.—Macroscopical Findings.—The macroscopical appearances are very characteristic. The liver may be considerably enlarged, especially in the earlier stages, or the organ may be about normal in bulk, while in some cases it is quite shrunken. It is usually deformed, and is roughened on the surface by nodules, which vary in size from that of a small pea to a hazel nut. These nodules, as seen through the capsule, are yellowish-brown, or chestnut-brown in color, the large ones often being bile-stained and dark. The spaces between the nodules are of a grayish translucent color, and may contain dilated veins. The entire organ is of a reddish-brown color, from which it derives its name, cirrhosis. The liver loses much of its friability, feels firm, and creaks when it is cut. The cut surface reveals nodules and a granular appearance, caused by the pressure on the lobules from the fibrous tissue. Sometimes the cut surface is universally dark brown from the presence of liver substance; but at other times the surface presents only patches of a brownish or greenish color, while the most of the surface is composed of solid elastic fibrous tissue. The cells may appear normal, fatty, pigmented, or necrosing. A close inspection of the cut nodules shows that they do not contain the normal number of lobules, and they do not bear their true relationship to the portal veins, the biliary ducts, and the hepatic veins. The lobules are not uniform in size and in many there is no central vein, the surface appearing smooth and granular. Parts of lobules, whole lobules, or groups of lobules may be destroyed. When the liver is small, the surface is more nodular than in livers that are larger, and merit the name "hobnailed." The organ varies greatly in weight, ranging all the way from 30 to 200 ounces, the average being about 10 ounces heavier than the normal liver. The term "hypertrophic" as applied in this disease, when the liver is large, is unfortunate and incorrect, for the enlargement is not a "hypertrophy," but merely an enlargement, and in those with large as in the case of those with small livers, the condition is essentially the same. Further, the use of the term "hypertrophic" in this connection leads to confusion with Hanot's "hypertrophic biliary cirrhosis," an entirely different disease. When the cirrhosis has been caused by alcoholic indulgences the liver tends to be larger than when the cirrhosis has been induced by other toxic irritants. Occasionally, extensive varices are found on the surface of the liver. The cut surface of the liver reveals a complicated system of connective tissue trabeculæ arising from the capsule and portal canals. The spaces enclosed by this fibrous arrangement contain a number of lobules.

Microscopical Findings.—The microscope reveals several changes in the histology of the liver. There is an increase in the fibrous tissue; a disordered arrangement of the hepatic cells; evidence of pigmentation, fatty degeneration, and necrosis going on in them; and an increase in the size and number of branches of the hepatic artery. There is also evidence of a distinct effort at the regeneration of the liver cells. In the normal liver the cells in the lobules are arranged in radiating rows, but in the lobule of a cirrhotic liver these cells become a jumble, their proper relationship to the portal and hepatic veins is lost, and there is only a conglomeration of cells and capillaries, through which the blood finds its way with difficulty. Around these areas is found vascular connective tissue containing many small cells, leukocytes, and tortuous bile-ducts. In this connective tissue may be seen the remains of hepatic veins, which drained the lobules. This new-formation connective tissue also separates the liver cells from the portal veins and the bile-ducts. The bile-ducts can be seen, and branching from them many new channels, lined with epithelium and connected with groups of cells. These are new bile-channels in formation. It does not appear that this connective tissue formation destroys the biliary cells, but rather that the injurious agent affecting the liver destroys its cells, and the connective tissue formation is reparative and secondary. It becomes clear that if the harmful agents acting upon the liver are removed, the liver cells can effect much restoration.

History and Distribution.—Cirrhosis of the liver has been known for a long time. A hardened condition of the liver was mentioned by Vësalius, Morgagni, and Harvey. Browne, in 1685, described a case and gave a drawing of the liver. Baillie distinguished cirrhosis of the liver from some forms of tumor disease. In 1819, Laennec used the term cirrhosis, on account of the color of the organ. At a later date Carswell, Kiernan, and Legg examined the histology of these livers.

It is a disease of very wide distribution and is not confined to countries using alcoholic drinks. Improper foods, certain infections, and irritating drink concoctions may cause it.

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HYPERTROPHIC BILIARY CIRRHOSIS

(*Hanot's Disease, Hanot's Biliary Cirrhosis, Lobular Cirrhosis, Biliary Cirrhosis*)

Etiology, p. 107—Symptomatology, p. 108—Diagnosis, p. 110—Complications, p. 110—Clinical varieties, p. 110—Treatment, p. 111—Prophylaxis, p. 111—Prognosis, 112—Pathology, p. 112—Historical summary, p. 113.

Etiology.—PREDISPOSING CAUSES.—*Sex.*—The disease occurs slightly more frequently among males than among females. The ratio is about 59 to 41 per cent. Among adults it is 85 per cent. to 15 per cent.

Age.—The disease is very rare after forty years of age, whereas in interstitial cirrhosis the average age is forty-eight years. The most usual age is thirty to forty, though it is met with in a fair percentage of all the cases among children.

Heredity.—There does not appear to be any evidence that the disease is hereditary. There are a few examples of two or more members of a family suffering from the disease, but this may have been due to a common cause in the environment. It has been argued that in some families there may be a liability to contract infections in the biliary structures, as some families are prone to bronchitis or rheumatism.

Alcoholism.—In hypertrophic biliary cirrhosis alcoholism occupies an unimportant part in the causation, and, at most, only acts by inducing gastro-intestinal irritation which favors the invasion of an infecting organism.

Association with Other Diseases.—Malaria, tuberculosis, and syphilis have not been shown to have any influence in the production of the disease. As the typhoid fever bacillus may set up a cholangitis, the theory has been put forth that this infection may enable some other organism to find its way into the finer biliary tubules. When one recalls how common typhoid fever is and how rare is biliary cirrhosis, it would seem that there was not much foundation for this view of its origin.

EXCITING CAUSE.—After giving due weight to the foregoing predisposing causes, one must look for some more direct explanation for the genesis of the disease than those mentioned. It must be admitted that there is present in the biliary tubules a persistent and chronic inflammation—a chronic cholangitis. This compels the consideration of an infective process. In support of this view may be mentioned the frequent manifestations of fever, the improvements and relapses, the glandular enlargements, and the morbid changes in the spleen. As to the route taken by the infection, two theories have been advanced. One of these is that it reaches the liver by the hepatic artery and descends from the smallest biliary capillaries. The other is that it enters from the duodenum and ascends to the finer tubules.

There are reasons for regarding both routes as possible. The presence or absence of gastro-intestinal irritation, and the precedence or sequence of splenic enlargement, as compared with that of the liver, give grounds for such a belief. In cases where the gall-bladder has been drained, the colon bacillus and the *Diplococcus pneumoniæ* have been found. The improvement that has followed this operation lends further proof to the infective theory. Both of these organisms have been found in blood drawn from the liver in biliary cirrhosis; but it cannot yet be said that they are the causative bacteria.

Symptomatology.—CLINICAL HISTORY.—*Period of Incubation.*—If this disease is admitted as due to some form of infection, the incubation stage varies greatly; in the majority, however, it is slow. There may be a lengthy period of ill health before any of the definite symptoms of the disease make their appearance. At this time the patient may complain of little else than indigestion.

Mode of Onset.—The actual onset of the disease is characterized by loss of strength, a feeling of indisposition to make an exertion, dyspepsia, itching of the skin, pain in the region of the liver, and jaundice. This latter condition is the one for which advice is first sought by the majority of sufferers.

Symptoms During Progress of Disease.—It is essentially a chronic disease. During the progress there may be periods of apparent improvement, to be followed by periods of marked exaggeration of the symptoms, with a downward trend in the condition of the patient. These periods become more frequent and more severe as the case progresses.

Objective Symptoms.—The tongue is frequently coated, and vomiting occurs; there may be hematemesis, especially toward the end of the disease. Diarrhea is fairly frequent and may be induced on very slight provocation. The stools are rarely clay-colored, as the jaundice is seldom so complete as to give rise to this feature. The abdomen is distended, especially in the upper part, due to the enlarged liver and spleen, and the tympanites; and the lower part of the abdomen may be distended with fluid. Enlarged veins may develop around the umbilicus, as in portal cirrhosis of the liver. Jaundice makes its appearance early in the disease and is permanent, though it varies in depth at different times, the color being sometimes light and then dark, but ever tending to increase until the end, the skin becoming even greenish, or very dark brown. There may be hemorrhages from the nose, the gums, the throat, the lungs, the kidneys, and the stomach. The patient becomes emaciated, and, if the disease begins in early childhood, the growth is considerably arrested, almost to the degree of infantilism. In chronic cases there may be observed an extreme degree of clubbing of the fingers and toes. The ends of the long bones and some of the joints may be seen enlarged. The breathing may be shortened and embarrassed due to the enlargement of the liver and spleen or the accumulation of fluid. The lower ribs may be bulged outward.

Subjective Symptoms.—The patient early complains of pain in the left hypochondrium, due to the increased size of the spleen. Pain may be felt rather widely throughout the abdomen. There is usually nausea and, at times, it may become extreme. There is a sense of weight and fullness in the right hypochondrium, and attacks of pain in the area of the liver and spleen. The toxemia may cause mental depression, emotionalism, and impairment of memory.

PHYSICAL FINDINGS.—By palpation and percussion the liver will be found to be evenly enlarged, and usually smooth on the surface. In those cases with a perihepatitis, however, it may be irregular in shape. The dullness may extend from the umbilicus to the fourth rib. The liver has been known to extend to the ilium. Palpation is likely to elicit general tenderness, but no acute local tenderness, and seldom any in the gall-bladder, which is not distended. The enlargement of the liver can be felt to undergo steady increase, but occasionally there is a shrinking in this regard, due to a true fibrosis.

The spleen is enlarged, firm, and smooth, and sometimes is larger than the liver, a condition which is more likely to be met with in children than among adults. Its enlargement may precede that of the liver. When the capsule of the spleen is inflamed, as it may become in these cases, a friction sound can be heard and a blowing bruit also is frequently produced in these engorged spleens.

Physical examination may discover an enlargement in the ends of the bones and a synovitis, which has been called biliary rheumatism. Cardiac murmurs may be heard, and, when they do occur, they are caused generally by dilatation. The pulse is often weak and irregular.

LABORATORY FINDINGS.—*The blood* in most of these cases is in a state of secondary anemia. The red corpuscles are reduced in number. The writer had one case where the count was 2,150,000; the reduction is more generally of a moderate degree. Emerson mentions a case where the count was as low as 1,504,000. He also reports 2 cases with a red count as high as 7,800,000 and 8,500,000. Hayem's case had 1,884,000 red corpuscles and 50 per cent. hemoglobin. Leukocytosis is a feature of the disease, and rarely rises above 12,000, but is oftener about 9,000. When leukocytosis is present the polymorphonuclears predominate. The blood has lost some of its coagulability, as might be expected from the jaundiced condition.

The urine in the majority of cases is increased, but may undergo reduction in the more acute phases of the disease. Bile-pigment is almost always present, and urobilin and indican sometimes. The urine contains less than the normal amount of urea, and this may become markedly decreased. There is no tendency toward albuminuria, and when albumin is present it is generally a temporary condition. Casts are frequently found in the urine. Glycosuria is absent, and a test dose, giving three ounces of sugar on an empty stomach, does not cause any to appear in the urine. The cryoscopic or freezing point of the urine is high.

SPECIAL EXAMINATIONS.—The employment of the x-rays may aid in determining the size of the liver and spleen. Information may be secured by the examination of a series of plates or by screening the patient.

Diagnosis.—The important features in diagnosis consist in the occurrence, in a young person, of jaundice, which runs a persistent course, and is accompanied by periods of exaggeration marked by increase in the jaundice, and the presence of abdominal pain; there is a negative history in the matters of alcoholic usage, gall-stones, tumors, or causes of biliary obstruction. Although the jaundice is prolonged and severe, there is an absence of the light-colored feces of other forms of jaundice.

Hypertrophic biliary cirrhosis must be differentiated from portal cirrhosis, hydatid disease of the liver, Banti's disease, tuberculosis and syphilis affecting the liver, obstruction of the biliary tubes, hemochromatosis, Weil's disease, malaria, and cancer of the liver.

In the case of *malaria* and *syphilis,* the discovery of the special organisms and tests, taken with the clinical history in each, should clear the ground. In *Banti's disease* there is a splenic anemia ending in hepatic cirrhosis and jaundice. In the case of *hemochromatosis* there is pigmentation rather than jaundice of the skin, and glycosuria in nearly all the cases, the cirrhosis of the liver and pancreas being a result of the condition. In *obstructive jaundice* there is the history of gall-stones with the severe pain of sudden onset, and the spleen is not enlarged. In *Weil's disease* the course is an acute one of days, not years. *Portal cirrhosis* is distinguished from hypertrophic biliary cirrhosis by the tendency in the former to early hemorrhages and, later, ascites, and the general absence of jaundice, or its manifestation in a slighter and transitory form, and the slight amount of splenic enlargement. In *hydatid disease* the spleen remains normal; there is an absence of periodic attacks of fever and pain, and jaundice is either absent or only occasional. Between biliary cirrhosis and *cancer* the points of distinction are that in the latter we find pain, loss of weight, failure of appetite, rapid and progressive enlargement with evidence of tumor, and jaundice coming on after the disease has made considerable progress, in a little over 50 per cent. of the cases.

Complications.—During the course of the disease there may be internal hemorrhages, but these are limited in amount compared with those occurring in chronic interstitial cirrhoses. Perihepatitis may also develop. Ascites may occur late in the disease. The most frequent complication is that of portal cirrhosis, from the toxemia of the disease. Cancer and biliary cirrhosis have occurred together.

Clinical Varieties.—Several varieties of hypertrophic biliary cirrhosis have been mentioned by different authors. These varieties all depend for their differences upon the ratio of size of the spleen and liver and the order in time in which the enlargement occurs. In the usual form of the disease both liver and spleen are proportionately enlarged, the change taking place at the same time. Then there are the *splenomegalic and hypersplenomegalic forms* in which the spleen leads the liver in rapidity of growth, and in the latter type becomes larger than the liver. The *metasplenomegalic form* is the one in which the enlargement of the liver follows that of the spleen, whereas in the *presplenomegalic form* the enlargement of the liver is the first. In the *heptomegalic form* the enlargement of the liver is much more prominent than is that of the spleen. *Microsplenomegalic* and *asplenomegalic* are terms used for those cases with small spleens. To those cases in which the liver is small or shrinks, the term *atrophic biliary cirrhosis* has been given. In some juvenile cases the spleen becomes very large. In the *metasplenomegalic form,* or the variety in which the enlargement of the liver follows that of the spleen, and appears to arise from poisons generated in the spleen, a very close alliance with Banti's disease is established.

Treatment.—PROPHYLAXIS.—Prophylactically little can be done, as the causes of the disease are still in doubt. The **avoidance of drinking impure water** and the regulation of digestive derangements might prevent some cases. The thorough care of the health in convalescence from infections is a wise precaution.

GENERAL MANAGEMENT.—*Diet.*—The **diet** must be **light,** easily digested and nutritious. Milk should form an important part of the dietary; bread, butter, light puddings, cereals, eggs, fish, fruits, vegetables, and some meat may be allowed, according to the fancy of the patient and the manner in which these foods agree with him. Stimulating foods and alcohol should be avoided.

Climate.—If possible the patient should move to a healthy locality; but, under any circumstances, he should leave the house and place where he resided when the disease began. A cold, damp residence should be avoided.

Treatment of Symptoms.—Of the symptoms that may call for treatment, *intestinal fermentation* is likely to claim attention. Many intestinal antiseptics have been recommended, such as **salol, beta-naphthol, bismuth salicylate,** etc. They are of very doubtful value. The **careful regulation of the bowels** will do much toward the prevention and relief of this condition, and **magnesium sulphate or sodium sulphate** singly or in combination are very useful correctives of constipation and fermentation. Rolleston regards **calomel** in small doses, grain 1/40-1/20 (0.0016-0.0032 gram) three times a day, as the most trustworthy remedy at our command. **Hexamethylenamin (urotropin)** in doses of grains 3-6 (0.195-0.4 gram) several times daily should be tried to relieve the *chronic inflammation* and, perhaps, *infection* in the biliary tubes, as this drug is eliminated by this route. **Sodium salicylate** is another drug that acts favorably on the biliary ducts.

Pruritus, caused by the jaundice, may become very troublesome, and require treatment. For this condition **antipyrin** has been recommended in doses of grains 5-10 (0.325-0.650 gram), or **calcium lactate,** grains 20 (1.3 grams) several times a day. **Warm baths or carbolic acid lotions** are useful. **Pilocarpin,** hypodermically, has been tried advantageously in a number of instances.

The discovery of the infecting germ and of a curative vaccine should be the aim of the future.

SURGICAL TREATMENT.—The gall-bladder has been drained in a considerable number of cases, with marked improvement in some and cures in others. With regard to the cases said to have been cured, there is reason to doubt the diagnosis. However, as the prognosis is very unfavorable, the operation is justifiable in most cases.

Prognosis.—The prognosis as to recovery is most unfavorable, but as to duration of life the disease is chronic, running a course of many years, lasting as long as ten years, and averaging four or five.

The mode of death is that of toxemia—exhaustion, delirium, fever, and coma. If the jaundice is severe the prognosis is proportionately grave; and complication with other infections, such as pneumonia, erysipelas, or peritonitis, seriously lessens the patient's chances.

While the disease often has periods of remission, during which the patient's strength is fairly well maintained, there is no real recovery of function. Relapses are certain to occur, ending in death.

Pathology.—MACROSCOPICAL FINDINGS.—The macroscopical appearance of the liver is that of much enlargement, especially of the left lobe. The color is a dark green. The surface is smooth, with fine granulation; and only in cases where there is also the common form of cirrhosis combined with the biliary form is there any hobnail appearance. Sometimes there is perihepatitis and adhesions to the diaphragm. The liver is firmer than the normal organ, and when cut the surface has a mottled look, owing to the fibrous tissue bands running through the gland. The gall-bladder is generally healthy, and the larger bile-passages are normal. There is no tendency to form calculi. The hepatic artery and the portal and hepatic veins reveal nothing abnormal. The liver may weigh from 80 to 160 ounces. In the portal fissure the lymphatic glands are considerably enlarged.

MICROSCOPICAL FINDINGS.—The microscopical appearance is that of a typical unilobular cirrhosis, contrasting in this regard with the multilobular cirrhosis of the portal form. In the late stage of hypertrophic biliary cirrhosis the unilobular and multilobular characteristics may be combined. In unilobular or biliary cirrhosis the connective tissue is fine, and the elastic tissue is a growth from the biliary sheaths. This fine connective tissue may invade the lobules and be distributed between the hepatic cells. In portal cirrhosis the fibrous tissue is coarser and more dense than in biliary cirrhosis. The small bile-ducts are surrounded by fibrosis, some of them are undergoing obliteration, and many of the biliary capillaries are filled with plugs of thickened bile.

There is an interesting arrangement of cells between the lobules. These cells are grouped in columns in the interlobular connective tissue in such a manner as to form channels, constituting new biliary tubules which have either no or very few elastic fibers. The liver cells maintain their normal condition until the disease is well advanced. There is evidence of considerable effort at cell regeneration.

The spleen is much enlarged, occasionally to six times that of normal, while the liver is very rarely more than three times its proper weight. In Milian and Landrieux's case the spleen weighed 94 ounces. Its capsule may show evidence of inflammation.

The microscope reveals a general fibrosis and a distention of the sinuses with blood. The malpighian bodies in the early period of the disease are congested, while in the later stage there is fibrotic atrophy.

In the duodenum there may be no morbid changes, though some observers have found a catarrhal condition in a considerable percentage of their cases.

The pancreas is seldom increased in size, but reveals some important histological changes. There is a fibrosis spreading from the ducts, and an increase in the cells lining these, with fatty degeneration in the acinal cells.

The kidneys may be enlarged, but they are usually of normal size.

All the organs and tissues of the body are bile-stained; the discoloration, of course, varies considerably in degree with the severity and duration of the case.

Historical Summary.—Requin, in 1846, noted the disease, and Todd described it in 1857, and Hayem in 1874. Hanot's thesis, in 1875, was the first clear exposition of the condition, giving it the name "hypertrophic cirrhosis with chronic jaundice." Kiener, in 1893, suggested that the malady be called Hanot's disease. Oertel and Meyer, in 1908, contended that there is not sufficient reason for regarding it as a separate disease, but as a modification of cirrhosis of the liver taken in a general sense. This position is ably disputed by Rolleston. The writer is quite in accord with the view that it is a disease entirely distinct from portal or interstitial cirrhosis, on the one hand, and obstructive cirrhosis on the other.

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NONSURGICAL DRAINAGE OF THE BILIARY PASSAGES

Origin of method, p. 114; Technic and apparatus, p. 115; Value in diagnosis, p. 120; Functions of gall-bladder and biliary tubes, p. 124; Criticisms of the Lyon method of duodenobiliary drainage, p. 125; Endorsements of nonsurgical duodenobiliary drainage, p. 133; Value in treatment, p. 140.

Origin of Method.—The late Samuel J. Meltzer[1] of the Rockefeller Institute published an account of his investigations on the effects of magnesium sulphate when applied locally to the mucous membrane of the duodenum. According to his observations this salt when thus employed caused Oddi's sphincter surrounding the lower end of the common bile duct to relax and enabled the muscle of the gall-bladder to contract more freely, a combination of actions which he called contrary innervation. It must be borne in mind in this connection that these structures are supplied by motor and inhibitory fibres from the vagus, and splanchnic nerves, which act antagonistically to each other.

Professor Meltzer said "In experiments with magnesium sulphate I observed that the local application of a 25 per cent. solution of the salt to the mucosa of the duodenum causes a completely local relaxation of the intestinal wall. It does not exert such an effect when the salt is administered by the mouth, that is, when it has to pass through the stomach before it reaches the intestines. The duodenal tube has, however, apparently reached an efficient practical stage. I make, therefore, the suggestion to test in jaundice and biliary colic the local application of a 25 per cent. solution of magnesium sulphate by means of the duodenal tube. It may relax the sphincter of the common duct and permit the ejection of bile, and perhaps, even permit the removal of a calculus of moderate size wedged in the duct in front of the papilla of Vater. Twenty-five c.c. of the solution as a dose for an adult will bring no harm. For babies the dose should not exceed 4 c.c. The procedure could be developed into a practical useful method." Dr. Lyon [2] remarks regarding Meltzer's statement as follows: "This study conducted for fifteen months in France on men of the U. S. Navy Base Hospital No. 5, for six months in the Clinic of the Jefferson Hospital, in the wards of the Methodist Episcopal Hospital and in the author's private practice, is a study which has now been made of more than 100 cases with a total of nearly 1,000 observations. We have proved and are now in a position to demonstrate that Meltzer's assumption is correct."

Technic and Apparatus.—Following these physiological observations of Meltzer, Dr. B. B. Vincent Lyon [2] of Philadelphia has made practical use of them, and has succeeded in applying topically to the duodenum a solution of magnesium sulphate through an Einhorn's [3] duodenal tube, and also in obtaining specimens of bile by means of the same apparatus. When the tube has reached the duodenum he introduces through it 75 c.c. of a 33 per cent. solution of the salt, and the contents of the duodenum are then gently aspirated. On this subject with regard to procedure [4] he writes thus:

"Before the magnesium sulphate has entirely run in, the tubing is connected to the bottle and gentle aspiration started, and the magnesium sulphate returns at first uncolored, but within one to six minutes, normally, the sphincter is relaxed and the magnesium sulphate becomes tinged with bile, which becomes steadily deeper until pure bile alone is being recovered. When the first bile, which I call 'A' bile, and believed to be that contained in the common duct, plus a few drops from the cystic duct and a few mils perhaps of freshly secreted liver bile passing down the hepatic ducts, deepens to a distinctly deeper golden-yellow or becomes in any way off color and of heavier viscosity, this bottle is detached and drainage of this darker bile allowed to continue until the third transition to a much lighter yellow and thinner bile appears, when a final bottle is attached to continue the bile collection to the end of the drainage period.

"The darker bile appearing in the second transition I will call 'B' bile, and believe it to be almost entirely from the gall-bladder, mixed, of course, with a few drops or mils of liver bile. . .The third type, of very light yellow limpid bile which appears in normal cases in the third transition, I will call 'C' bile and believe it to be bile recently elaborated by the liver cells and freshly secreted. I feel safe in saying that, if carefully done, the majority of 'A' bile is common duct bile, that by far the majority of 'B' bile is delivered from the gall-bladder, and if the latter has emptied completely, that practically all of 'C' bile is freshly secreted liver bile.'' Einhorn's objections will be discussed later in this article. With regard to the technic and the apparatus a few words are now in order. Gross[5] gives the following method:

''TEST MEAL.—The patient is given the morning tumbler (250 c.c.) of milk and water, equal parts. This mixture does not cause a great flow of hydrochloric acid and contains sufficient fat to stimulate the pancreatic secretion. Half an hour later introduce the duodenal tube.

''TECHNIC.—The patient swallows the ball and tube, previously wet in water, until the mark 45 cm. reaches the lips. Then blow lightly through the tube into the stomach, so as to smooth the tube out and that it will lie freely in the cavity. The patient should then lie down and slowly turn over on the right side, in which position, after a few minutes, the tube is allowed to glide down through the mouth without swallowing, following the pull of the ball, until the mark 60 cm. has been reached. After five or ten minutes the first aspiration may be made, which usually shows contents of a slightly yellow tint. The patient, with the mouth still partly open, should not make any swallowing movements. Gradually the tube will be drawn down to the 65 or 70 cm. mark.

''A second aspiration is then made, and usually a yellowish fluid, free from casein, will be aspirated, giving a weakly acid or acid reaction. One should wait a little longer and, after several aspirations, the yellow aqueous contents of the duodenum will usually be secured, giving a neutral or alkaline reaction, and at times a greenish-yellow fluorescence.''

Dr. Lyon[2] has given a very explicit account of his methods as follows:

''The patient is examined in the fasting stomach state; the mouth is rinsed thoroughly with any good antiseptic solution. We prefer potassium permanganate, 1 grain to 2 ounces of water, followed by a rinse with a weak solution of zinc chlorid. A sterile duodenal tube, fitted with any one of the later modifications of the original metal tip, is passed into the stomach; the fasting gastric residuum is aspirated into a sterile vessel, is measured and grossly observed for consistency, mucus, etc.; and then is cultivated and studied chemically for acidities, for bile and for occult blood. Microscopically, we pay special attention to endogenous epithelium, to polymorphonuclear leukocytes, with or without digested protoplasm, and to the general amount and the morphology of the bacterial flora, differentiating between these and the findings which are similar but of exogenous origin. With care and the enlargement of personal observation, this differentiation can be made with a reasonable degree of certainty, and it is later checked up by other methods. The stomach is then thoroughly rinsed and the patient is given a glass of water to drink while slowly swallowing the tube to the duodenal point, and while lying on the right side, with a pillow or sandbag elevating the hips.

"The tube usually passes into the duodenum within fifteen to forty-five minutes. When it has done so the fact can be determined by the duodenal 'tug,' the character of the aspirated fluid, and the failure to recover immediately by vacuum aspiration material (water, broth, etc.), which the patient swallowed by mouth. Difficulty is occasionally encountered in entering the duodenum because vagotonic states, or local pylorospasm states (reflex from duodenal ulcer, cholecystitis, or chronic appendicitis), which can be overcome by an injection of atropia sulphate or by several days' use of belladonna or benzyl benzoate. Once it is sure that the tube is in the duodenum, a barrelful of air is introduced from a one ounce capacity syringe to balloon out the duodenal walls from the metal tipped tube (to prevent traumatism of the duodenal mucosa); a connection is made with the first sterile aspirating bottle, and gentle aspiration of the duodenum begun.

"On withdrawing the tube, a resistance may be felt at the pyloric ring which can be overcome without difficulty. A constriction at the esophagus will be overcome by a few movements of deglutition."

In his article on the use of the tube, Dr. Gross[5] lays down these precautions: "Occasionally the aspirated fluid remains acid, probably due to hyperchlorhydria, and, therefore, neutralization of the duodenal contents may occur lower down. As a check, give a cup of coffee with the tube in place. Aspiration should be performed, and if the fluid is still green, the contents are duodenal. This may be proved by withdrawing the tube a short distance and reaspirating, when the coffee will appear." Gross holds that unless there is a mechanical obstacle at the pylorus, it is usually possible to pass through the pylorus with the tube and into the duodenum within an hour.

"The Gross tube[6] consists of a perforated round metal ball about twice the size of a pea, to which is attached a thin rubber tube 0.2 cm. in diameter and 125 cm. in length, marked every 10 cm. To this is attached a glass receptacle (bulb), which is connected by a length of tubing with a mouth piece which the operator can use to aspirate, or to which an aspirating bulb can be attached."

Dr. L. Winfield Kohn[7] has devised a duodenal tube with some interesting new features. The following description is taken from his published account:

"This instrument consists of hard rubber stop cocks, a silver Y, rubber tubing, size 16 or 17 French, perforated olive body with a double channel, inner tubing of 12 French size, olive weight brass tip, 23 French size, thin tissue or glove rubber (for air bag), with fine silk chiffon surrounding it as a retentive or supportive agent. The rubber air-bag, when it is blown up, should be circular, or as near circular as possible. The rubber connections are securely tied with waxed silk thread. The instrument must be practically airtight or else the air-bag will quickly collapse. One avenue communicates with the air-bag which is blown up and serves as a blockade. The other avenue carries the shadow producing medium."

Through the avenue which carries the shadow producing medium, the magnesium sulphate solution can be injected, and the bile or other fluid contents in the duodenum aspirated.

Dr. Max Einhorn[3] has given much study to the perfection of the duodenal tube, and has devised a number of modifications of the instrument. The following gives the general features, and makes it clear how solutions can be placed within the duodenum, or fluids found therein withdrawn.

He describes in his book on The Duodenal Tube a method of passing a catheter through the pylorus. A buckshot is attached to a thread and swallowed. By carefully adjusting the position of the patient the shot passes through the pylorus into the duodenum. The end of the thread outside the patient's mouth is passed through an opening in the end and emerging at the side of a catheter of sufficient length, which is then passed down the esophagus, along the stomach, and through the pylorus guided by the thread held in the operator's hand. The markings on the thread and catheter indicate when the shot and the catheter have entered the duodenum. On the lower end of the catheter may be attached a balloon for the purpose of dilating the pylorus.

He also describes a tube with two perforated capsules, one for the level of the stomach and the other for the duodenum. By this two avenue tube the contents of the stomach and duodenum can be aspirated simultaneously.

"PREPARATION OF PATIENT FOR DUODENAL EXAMINATION.—It is best that the patient should take only fluid food on the day of examination. In most cases I have had him take a cup of tea and sugar without milk half an hour before examination.

"Lately I have used clear bouillon (one bouillon cube to a cup of hot water) for the same purpose.

"MODE OF PROCEDURE.—The capsule of the duodenal pump, as well as the lower part of the rubber, are moistened with warm water and put into the pharynx of the patient. Then the latter drinks some water, and the instrument thus soon passes into the stomach. To be certain that the capsule did not get stuck in the esophagus, it is well to have the patient shake his abdomen and to aspirate a syringeful of chyme. This can easily be identified as gastric contents. Now we pass a syringeful of water and then one of air through the instrument. The rubber tube is then clamped off and left alone for about one hour. The patient is told not to close his mouth too tightly, so that the tube is not retarded in its wanderings. The patient must also avoid intentional swallowing of the tube. Through the peristalsis of the stomach the capsule is pushed on further, and usually passes through the pylorus into the beginning of the small intestine. It is best to have the patient read some light literaure, in order to divert his attention. After one hour we examine how far the capsule has progressed; if sign III is near the lips (70 cm.), or inside the mouth, we try to aspirate. If the capsule is in the duodenum, we gradually obtain a clear, golden-yellow or watery liquid of alkaline reaction and somewhat viscid consistency. If, however, we are in the

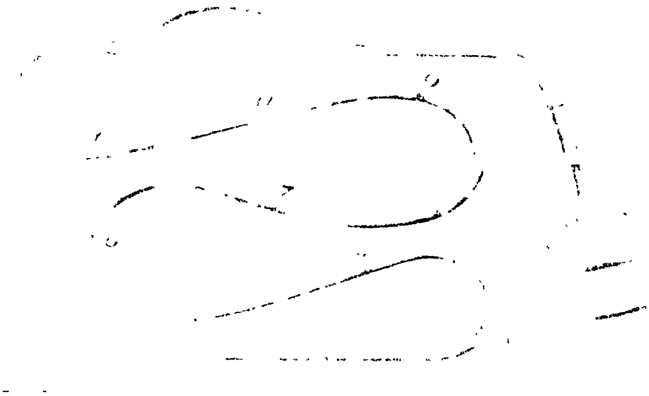

FIG. 1.—THE GROSS TUBE

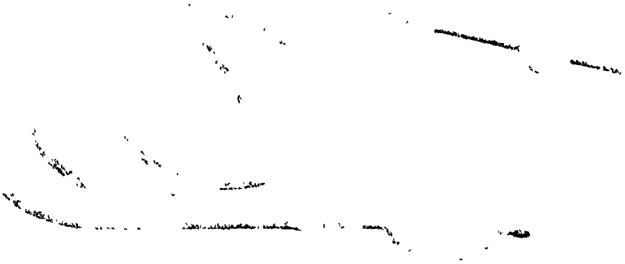

FIG. 2.—L. W. KOHN'S DUODENAL TUBE (MEDICAL RECORD).

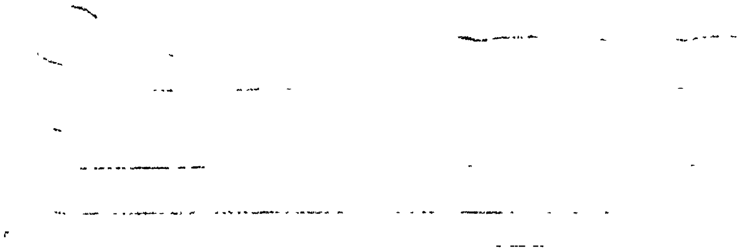

FIG. 3.—EINHORN'S DUODENAL PUMP.

(*a*) Metal Capsule, lower half provided with numerous holes, the upper half communicating with tube *b*. I, II, III, marks of 40, 56 and 70 cm. from capsule, (*c*) rubber band with silk attached to end of tubing, which can be placed over the ear of the patient; (*d*) three-way stopcock; (*e*) collapsible connecting tube, (*f*) aspirating syringe (from Einhorn's "Duodenal Tube").

stomach, we obtain an acid liquid resembling the one first removed. This can, of course, occur if the tube lies in the stomach in a coiled manner. Should the latter be the case, we must partly withdraw the tube, after putting water and air through it, up to the mark II (56 cm.). The tube is then again clamped off and after one-half to one hour the procedure is repeated. We generally succeed in nearly all cases (of course, not in such where there is pyloric stenosis) to obtain duodenal contents. After having obtained the desired contents, the tube is clamped and slowly withdrawn. When the beginning of the esophagus is reached the patient is told to swallow, and during this act the capsule is withdrawn."

Occasionally much difficulty is experienced in passing the tube through the pylorus. The following rules will materially assist the operator:

If the tube does not enter the duodenum by the end of three hours the patient lies on his right side for half an hour when aspiration is performed.

If gastric contents are obtained, the patient is given something to drink and told to walk about for 10 or 15 minutes. If the examination shows that the tube is still in the stomach some Vichy water, or drink containing carbonic acid gas is given. In half an hour aspiration is again performed.

If the tube has still failed to enter the duodenum, the patient is given atropin sulphate, grain 1/120; and in one hour later aspirate again. If stomach contents are still obtained, an aperient of magnesium sulphate is employed.

If the tube does not pass into the duodenum there is most probably pyloric stenosis, and an operation may be demanded. In such cases the tube can sometimes be passed by resorting to the use of the bead and thread.

A bead, size 14–15 F., is wrapped up in gauze and attached by a braided silk string (English No. 5), about 20–25 cm. long, to the end of the tube. This can be done by pulling the string through the lowest two openings in the capsule and fastening securely.

The bead is now placed in the pharynx and by drinking water made to enter the esophagus. The capsule follows into the pharynx and is taken down the esophagus by swallowing some water, and soon reaches the stomach.

As the bead is smaller than the capsule, it may pass through the pylorus, the string dragging the capsule after it. Lying on the right side or walking aids this.

George E. Brown [8] outlines his methods as follows: After a meal of shredded wheat biscuit and 10 ounces of water, we introduce a Rehfuss stomach tube. Aspiration is performed every 15 minutes until no food is found. The patient then turns on the right side with the left knee flexed over the right. The tube usually enters the duodenum in less than an hour, and this is known by:

(1) The change in reaction when the gastric fluid is acid.

(2) The duodenal juice is usually limpid, alkaline, with floccules in the fluid, and bile may be present.

(3) The presence of a slight tug, detected on gently pulling the tube.

(4) The x-ray revealing the bulb in the duodenum.

It was tried in 125 cases and in over 200 observations. The normal duodenal juice is alkaline in reaction, and is a pearly fluid, with viscosity less than that of the gastric juice. Floccules are seen suspended in this fluid. On microscopic examination a few microorganisms are seen and a very few cellular elements.

In pathologic conditions in which the duodenum is involved as a part of the infection, the duodenal fluid is turbid and is loaded with microorganisms with an increase of mucus; a bile admixture is frequently found.

After being assured that the bulb is in the duodenum, we slowly inject 50 c.c. of sterile magnesium sulphate solution. This is injected warm, by means of a 50 c.c. syringe. After a few minutes a light yellow bile appears in the syringe, gentle suction having been employed. We then disconnect the syringe and allow the tube to hang over the side of the bed, draining by gravity. The first bile appearing is an admixture of duodenal fluid, common bile-duct bile, and magnesium sulphate solution after douching the duodenum mucosa. After a variable period of from ten to thirty minutes, the bile becomes darker in color and more viscid. This is the gall-bladder bile and is collected in separate sterile tubes for study. The amount varies from 25 to 75 c.c. When there is an excess of 80 c.c. we consider the gall-bladder as presenting the condition of stasis. Finally the bile becomes lighter in color, a clear light yellow, less syrupy, and this is the liver bile, which may continue flowing for an indefinite period. The common duct bile is usually small in amount and clear (with perhaps a trace of mucus). The normal gall-bladder bile varies from a deep golden-yellow to a dark maple-brown; it is moderately viscid, dropping easily in short lengths from a 1 c.c. pipette; it is clear. Small traces of mucus may be seen in apparently normal gall-bladder bile, but this must be viewed with suspicion. Its reaction is neutral or faintly alkaline. Its specific gravity varies from 1.015 to 1.030; this may be increased to 1.035 in cases of biliary stasis and infection. Liver bile is thinner, and the specific gravity is from 1.008 to 1.010.

Value in Diagnosis.—With regard to the diagnostic value of the duodenal tube Dr. Lyon[9] puts the case thus:

"In pathological states of the gall-bladder, the appearance and the character of the bile are quite different from those in the normal bile. We have seen the following types:

"(*a*) *Choledochitis.*—In choledochitis the bile first collected is definitely pathogenic. It is viscid, with an excess of flaky mucus, is usually turbid and usually 'off' color; cytologically, it contains pus cells enmeshed in mucus, epithelial cells, and, occasionally, red blood corpuscles; bacteriologically, cultures may show pathogenic organisms. This type has been encountered in choledochitis with cholecystitis.

"(*b*) *Cholecystitis.*—In cholecystitis without choledochitis, the first bile collected is relatively normal. Grossly cytologically and culturally it does not contain pathogenic organisms. But the second bile is grossly pathologic, it is more viscid than normal gall-bladder bile; it is turbid, with a flaky or stringy mucus; cytologically it will show inflammatory elements, pus cells, red blood-cells (occasionally), and desquamated epithelium; and cultures will show pathogenic organisms, such as streptococci of various strains, bacillus pyocyaneus, bacillus typhosus, and micrococcus catarrhalis. The viscosity, color and general appearance of the bile in pathologic conditions of the gall-bladder will alone suggest the diagnosis. The color varies from a deep, golden-yellow to a dark molasses yellow, transparent or turbid; to a light, mustard-yellow, always turbid, and occasionally streaked with slimy mucus of a greenish color; to various shades of green or greenish-black, and, in one case, was of such tarry consistency as to make aspiration very slow and difficult.

"(*c*) *Cholelithiasis.*—In cholelithiasis we find the evidence of cholecystitis, but in two instances we have noted in addition that the bile contained a sediment that was 'gritty' or sandlike in consistency, which was seen, microscopically, to be made up of crystals of bile salts. In one case three small gall-stones were recovered. So far no example of infected bile had been discovered coming direct from the liver, after all the bile in the ducts and gall-bladder had been removed."

The causes of infection are: (1) There is infection by the ascending route from the duodenum by way of the common duct.

(2) Infection through the portal vein and possibly through general circulation.

(3) As to the first route the sequence of events are often as follows: infection from gums, teeth, nasal sinuses, the respiratory tract, as in pneumonia and bronchial disease, which infections are swallowed, setting up disease in the stomach and duodenum, and thence ascending the common bile duct.

Securing Duodenal Contents.—It is important that in the various steps taken to obtain the contents of the duodenum a proper technic should be followed. This may best be given in Dr. Lyon's own words.[10] This is the more essential as a similar procedure should be followed in the treatment of patients by means of the duodenal tube.

"To make possible accurate diagnosis of the duodenal biliary zone it is necessary that we adopt means to prevent cytological and bacterial contamination from the mouth, teeth, tonsils, respiratory tract and stomach from confusing us in our interpretation of duodenal and biliary materials. To avoid this as far as possible I have adopted the following routine method: The patient presents himself with a twelve-hour fasting stomach. He then brushes his teeth carefully, rinses and gargles his mouth and throat thoroughly, first with a strong solution of potassium permanganate (one grain to the ounce), then with a mildly astringent solution of zinc chlorid. The duodenal tube which has stood over night in a two per cent. solution of lysol is freshly sterilized by boiling and is passed to the stomach. The fasting residue is aspirated and set aside for chemical, cytological and bacteriological examination for comparison with the findings later recovered from the duodenum. The stomach is then rinsed to sparkling clearness, using gravity douching from 250 c.c. irrigating tank or syringe, douching and recovering the wash-water in 250 c.c. conical graduates in which can be noted how clean the stomach is, mucus, shreds, mucopurulent plugs, and other material which microscopically yields valuable information. After the wash return is sparkling clear the stomach is made astringent with a zinc chlorid solution and then rewashed thoroughly.

"It is surprising how often a stomach apparently washed clean, after being made astringent, will press out from the mucosal tubules mucopurulent masses which plug the ducts and which microscopically show, in true gastric cases, masses of gastric epithelial cells infiltrated with small round cells and polynuclear leukocytes and often swarming with bacteria. It is to be noted that none of this epithelium is ever bile stained. After the use of the astringent and washing clean again, the stomach is then disinfected with 250 c.c. of potassium permanganate, one to ten thousand, which is immediately recovered and the stomach again washed clean to crystal clearness. This requires about twenty minutes to accomplish. This, so far as it is possible, prevents contaminated material from the upper zones confusing our interpretations of material later obtained from the duodenal biliary zone.

"The duodenal tube is then connected to the first sterile aspirating bottle and the duodenal secretion is aspirated to note whether the common duct sphincter is normally closed. The duodenum is then douched with about 75 c.c. of a 33 per cent. solution of magnesium sulphate. This is the optimum strength for good sphincter relaxation and gall-bladder contraction. Where the gall-bladder is found atonic it is sometimes necessary to restimulate its contraction discharge by douching again with half the amount of magnesium sulphate solution."

Following this procedure, whether for diagnostic or therapeutic purpose, Lyon recommends that the duodenum be given an enema of 250 c.c. of Ringer's solution, for its healing qualities, reinforced by a .5 per cent. or a .25 per cent. sodium sulphate, depending upon how much magnesium sulphate solution has failed to be recovered.* The total amount of the duodenal enema is kept at 250 c.c. introduced at a temperature of 105° F. (40.5° C.), taking at least twenty minutes. This quantity of fluid usually produces a large bowel movement in less than ninety minutes.

Examination of the Duodenal Contents.—Lyon[11] in 93 cases found that of those with bacteria present, 50 per cent. were of the streptococcic group, 25 per cent. of staphylococcic group, bacillus coli in 15 per cent., bacillus subtilis in 8 per cent., bacillus pyocyaneus and bacillus typhosus each 1 per cent. The color of the "B" bile varies much, but is darker than the "A" bile from the common duct, and the "C" bile from the liver, after the gall-bladder has been emptied. In one case [12] the bile was very dark, very tenacious, cloudy and was a deep green-black. On the second use of the tube and magnesium sulphate, "the bile flowed immediately but was not so dark." The bile became progressively thinner and lighter in color as the drainage was repeated daily. The bile was positive for typhoid bacilli. In another case [13] the tube recovered over 5 ounces of deep greenish-black bile, very viscid, almost tarry.

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* Ringer's solution is a physiological salt solution of sodium, potassium and calcium chlorids, and sodium bicarbonate. Locke's modification of this solution and known as artificial serum is as follows: Sodium chlorid, 0.9 per cent.; calcium chlorid, 0.24 per cent.; potassium chlorid, 0.01 to 0.03 per cent.; and dextrose, 0.1 per cent.

Lyon [14] states that in the examination of the types of bile the following should be noted: Their gross appearance, color, consistency, viscosity, transparency, turbidity, flocculation, mucus, etc.; the cytology, as pus, leukocytes, crystals, inflammatory débris, mucus, red blood corpuscles, concretions, bacteria, and epithelium, its source and whether bile stained; the chemistry as lecithin, cholesterin, calcium, pigments, and effervescence on acidification.

The bacteriological examination should be made without delay to prevent streptococci and other less hardy organisms becoming overgrown with the more rapidly growing colon groups, bacillus pyocyaneus, and bacillus subtilis. All instruments and media should be thoroughly sterile. The best results are obtained by cultures made from the mucopurulent flakes, especially if bile stained; and they show the most conclusive cytological findings.

Frank Smithies [15] lays down these conditions as causes why aspirations for bile may fail: (*a*) Marked pyloric or duodenal spasm; (*b*) occlusion of the bulb perforations; (*c*) kinking of the tube; (*d*) the bulb too far from the papilla of Vater; (*e*) failure of magnesium sulphate to arouse duodenal reflex due to faulty innervation, atrophic duodenitis, backflow of magnesium sulphate into the stomach, its rapid flow down the duodenum; (*f*) occlusion of the papilla of Vater or the biliary ducts from thick mucus or bile, calculi, inflammatory products, kinks, new formations, adhesions, and external pressure; (*g*) to inhibitory or secretory reflexes not yet understood. On second and third attempts on these groups the unsuccessful aspirations were reduced to 4 per cent.

In a study of 1,000 cases of diseased gall-bladders he found that 63 per cent. contained viable bacteria. In the remaining 37 per cent. "other evidences of disease were found such as an abnormal quantity of gall-bladder bile, ranging from over 100 c.c., the normal, to 2,800 c.c. and averaging 292 c.c. The gross appearance was abnormal as bloody, dark-colored, turbid, thick mucoid bile of high specific gravity, with heavy, sometimes gritty, sand-like sediment, and small calculi may be found. On microscopic study there are found pus, desquamated, flat-columnar epithelium, excess cholesterin, soaps, bile salts and mucus."

Franklin W. White [16] does not attach much importance to the color changes, as the "A" bile may be mixed with "B" biles and "C" biles, and so may "B" bile be mixed with "C" bile. He thinks it is rare to secure pure specimens from the fractional portions of the biliary apparatus. He does think, however, that absence of any color change is important as indicating an obstruction in the cystic duct, preventing the appearance of the shades of bile. In examining for cells the preferable way is to pick up the shreds and flakes in a pipette, not using the centrifuge, and placing these on the slide for microscopic study. "In pathological cases there may be abundant deeply bile-stained pus cells and desquamated columnar and round epithelium, mucus, and rarely, blood-cells." In normal cases there is "a scanty biliary sediment, a few leukocytes and round columnar cells, a few fat needles or fat drops, and granular bile salts." Crystals and amorphous forms of cholesterol and bile salts "may be very abundant, suggesting the possibility of stone formation. There may be a fine gritty sediment of bile salts. The upper portion of the small bowel in fasting persons is usually sterile; and bile obtained by the tube yields results of considerable value; about one-fifth of the cultures were sterile, a few showed bacillus porteus and subtilis, staphylococcus albus. In some cases pathogenic organisms were found as streptococcus hemolyticus or staphylococcus aureus, matching with organisms found in the tonsil, sinus, or tooth abscess." In acute and subacute cholecystitis, the cultures were more frequently positive. The bacteria obtained by the duodenal tube corresponded fairly closely to that obtained from the gall-bladder at the time of operation.

Functions of the Gall-bladder and Biliary Tubes.—For a proper understanding of the non-surgical drainage of the gall-bladder and biliary passages a careful study of the functions of the biliary apparatus is essential. Towards the clearing up of this subject there has been much investigation and, while every phase of the case has not been fully explained, a number of questions have been settled. These are:

(1) That there is in the gall-bladder a small amount of muscular tissue [17] of the non-striated variety. In the normal healthy condition this is capable of contraction, which would have the effect of emptying the gall-bladder through the cystic and common bile ducts into the duodenum. The progress of digestion is the chief stimulus to this action. The non-distended bladder can hold 50 c.c., but in health can enlarge [18] without causing discomfort, so as to hold 200 c.c., or one-fifth part of the total secretion for 24 hours. It is clear, therefore, that it is a retaining sac for the accumulation of bile during remissions in active digestion. Three times this amount would be one-half of all the bile secreted in 24 hours. The gall-bladder can be and is a real storehouse for the bile in the healthy individual partaking of food at the usual intervals. The contractile power of the gall-bladder has been estimated to be about 300 mm. of water, while the sphincter at the papilla of Vater closes with at least twice this force. It seems plain that the gall-bladder in itself could not overcome the sphincter control; but the peptones of digestion act as a powerful stimulus to effect relaxation of the duodenum and the Oddi muscle, and permit of the free flow of bile from the viscus. It must be apparent to all that the bile in the gall-bladder does not remain there quiescent but must be undergoing frequent change. Passing over the views advanced that the mucous membrane of the gall-bladder forms a

hormone that stimulates the flow of bile and pancreatic juice, there is no doubt, as Rous and McMaster[19] and Harer, Hargis and Van Meter[20] have shown, but that the gall-bladder is a concentrating organ, and that cystic bile is on the average eight times as rich in solids as that from the hepatic ducts. These investigators have shown that in 22 hours the gall-bladder concentrated 49.8 c.c. of bile down to 4.6 c.c.

(2) The biliary passages appear to have functions other than that of carrying the bile. The hepatic duct conveys the bile from the liver to the point where it joins with the cystic duct to form the common duct. The cystic duct carries bile either from the above junction to the gall-bladder, or from the gall-bladder to the origin of the common duct. The common duct carries both the cystic and hepatic biles to the duodenum. Just as it is about to enter the duodenum it is joined by the duct from the pancreas. It has been well established by Judd and Mann[21] that after the removal of the gall-bladder all the ducts outside the liver dilate, but that this dilatation is most marked in the common duct where it approaches the duodenum. This is an effort on the part of nature to furnish a substitute for the lost gall-bladder as an adjuster of the pressure in the biliary tubes, and as a storehouse for bile not at the moment required.

(3) McWhorter[22] has recently carefully studied Oddi's muscle and finds that though there is not a complete muscular ring around the end of the common duct, there is an efficient sphincter action. He also found that it was difficult to overcome the tone of this muscle by incision without going through the thickness of the wall. On the other hand he found that it relaxed under the local action of a 25 per cent. solution of magnesium sulphate. He also observed that when the gall-bladder was removed the tonicity of the sphincter was much lowered, showing a relationship between this muscle and the gall-bladder.

(4) The cystic duct, as shown by Rous and McMaster[23], possesses a marked tendency both in dogs and man to redevelop a gall-bladder in cases of cholecystectomy. This is particularly the case where a considerable portion of the cystic duct has been left. Their words are: "Both in the dog and man a new gall-bladder may develop from the remnant of bladder mucosa left by a cholecystectomy that has failed to include the cystic duct." This power is also described by Hartman and Smyth[24], who also point out the dilatation of the biliary tubes after removal of the gall-bladder.

(5) In the observations of Rous and McMaster it was clearly demonstrated that the biliary tubes did not concentrate the bile, but on the other hand added water to it and diluted the bile, giving rise to "white" bile. When the obstruction is low down in the common duct the water from the biliary tubes more than counteracts the concentrating power of the gall-bladder, and the total bile becomes thinned and lighter in color. This is a very important function and should be kept in mind in dealing with gall, biliary and biliary duct problems.

Criticisms of Lyon Method of Duodeno-biliary Drainage.—A number of observers have at various times adversely criticized the method of non-surgical drainage of the gall-bladder and bile ducts advocated by Lyon on the suggestion of Meltzer. The most prominent of these opponents are Einhorn, Dunn and Connell, Crohn, Reiss and Radin, Bassler, Luckett and Lutz, Cook and Wilhelm.

The objections to this method of gall-bladder drainage advanced by these investigators shall now be taken up seriatim, and their position regarding its utility stated.

Cook and Wilhelm[25] state that their experiences do not encourage them to place much value in the Lyon method as an aid in diagnosis. There is, in their opinion, too much doubt as to the source of the three colors of bile.

Dunn and Connell[26] came to the following conclusions as the result of their study of a case of hepatoduodenostomy. In this patient the gall-bladder and common bile duct had been removed and the hepatic duct anastomosed to the duodenum. There was a fistula leading to the duodenum through which a catheter could be introduced for the purpose of collecting the bile that came directly from the liver.

(1) Eight days after the hepatoduodenostomy they introduced through a catheter into the duodenum 50 c.c. of a 30 per cent. magnesium sulphate solution, recovering the "A," "B," and "C" bile.

(2) In the morning, with the patient fasting, bile was collected in fractional portions over a period of two hours, no magnesium sulphate solution having been given. All portions of the bile were pale yellow and uniform in color and consistency.

(3) Into the colon per rectum there were injected 300 c.c. of a 30 per cent. magnesium sulphate solution. The bile collected in fractional portions showed no change in color or consistency.

(4) Through the duodenal fistula a catheter was passed 32 cm., and 100 c.c. of a 30 per cent. magnesium sulphate solution inserted into the jejunum. The fractional "A," "B," and "C" biles were obtained.

(5) In this experiment the procedure was the same as in No. 4, but the magnesium sulphate solution was injected in successive portions. The "A," "B," and "C" biles were obtained.

From these experiments they conclude that the changes in color in the bile are due to the action of the magnesium sulphate on the liver, and not to bile coming from different parts of the biliary passages. When the solution is injected into the colon it does not appear to reach the liver so as to influence the color of the bile; but does do so when injected into the duodenum or jejunum. It must be borne in mind, however, that the biliary tubes dilate soon after the removal of the gall-bladder. In this case they may have done so before the operation.

The magnesium sulphate solution in the upper portion of the small intestines secures the three color types of bile; whereas, when the solution is placed in the colon or taken into the stomach this does not occur. In Experiment 2 where the magnesium sulphate solution was not employed the bile did not change in color. This might have been due to the fact that the duodenal muscle was not relaxed and the bile flow was steady and mainly from the liver, the dilated tubes not emptying their contents with sufficient rapidity so as to yield the darker bile at one time, but gradually mixed with what came from the liver, obviating any observable change in color. It has been observed that the magnesium sulphate solution relaxes the duodenum and would in this case favor the flow of bile into it, although there was no sphincter of Oddi present. The sequence of events would be that the duodenum became relaxed; the hepatic duct emptied its contents of light color into the duodenum, then the bile that had accumulated in the dilated biliary tubes and of dark color, and, finally, bile fresh from the liver and light in color.

But as against these explanations for the color changes they say: "Chemical analysis showed that with deepening color of the bile there was a slight increase in its magnesium sulphate content, which must have been absorbed by the portal vein radicles, and resecreted by the liver with increased bile pigments and salts."

In a discussion of this subject by Bassler, Luckett and Lutz[27] a number of objections are advanced to show that the changes in the color of the bile cannot be accepted as safe guides towards a diagnosis:

(1) The first objection is that on visual observation and by palpation there was no contraction on the part of the gall-bladder. Four of the cases were watched by Bassler and six by Luckett. The patients were under a general anæsthetic, the abdomens were opened, and the intestines and gall-bladder were manipulated. Under these conditions the injection into the duodenum of magnesium sulphate solution through a tube, inserted prior to the anæsthetizing of the patients, failed to cause any contraction of the gall-bladder. When, however, the bile was expelled from the gall-bladder by gentle finger-pressure it was of the characteristic "B" type, such as is obtained by the tube from patients not under an anæsthetic. But in four of Luckett's six cases the second or third aspiration brought up from the duodenum darker bile than the first. This points strongly to an effort on the part of the gall-bladder to empty itself, an effort which was partially successful.

Here are a number of factors that might modify the action of the magnesium sulphate solution. In the first place the patients were under an anæsthetic which might lessen or abolish the contractible power of the gall-bladder. In the second place it might render the duodenum nonresponsive to stimulation by the solution, and in the third place there was the multiple effects of traumatism from the operation and the exposure to the air, effects which the utmost care could not obviate or wholly control, and might restrain contraction of the gall-bladder without arresting the peristalsis of the duodenum.

(2) These observers do not think any value can be attached to variations in the specific gravity in the bile from different parts of the biliary apparatus. They state: "We believe that the specific gravity practically corresponds to the salt content present in the specimen, and, further, that in gall-bladder biles not obtained by this method the specific gravity is not much elevated from heptic bile, at least to no practical degree, by the addition of the mucin it gets in the gall-bladder. This seems difficult to believe, but it is, nevertheless, true."

This position is contrary to well-known facts. The chemical composition of gall-bladder bile and liver bile differ very materially; and we have the carefully conducted experiments of Rous and McMaster, Harer, Hargis and Van Meter, and many others, proving that the gall-bladder possesses the marked power of concentrating the bile. Indeed, Rous and McMaster have shown that this may be to the extent of ten parts reduced to one part, thereby deepening the color and raising the specific gravity. According to Yeo and Herroun and Hoppe-Seyler the solid constituents in liver bile amount to 1.3 per cent., and in gall-bladder bile to 8.19 per cent.

Bassler and his associates say, "It is agreed upon by all that the bile contained in the gall-bladder is deeper in color than that which flows directly from the liver substance, and it is assumed by many that this change in color is due to concentration." It is a familiar fact that liver bile always contains some coloring matter, and is normally of a light lemon tint. Condense such bile into one-tenth its volume, as has been shown to be the case, and the color must be greatly deepened without the necessity for calling in the oxidation as an explanation.

On the view long held that the gall-bladder is a reservoir these writers remark, "It never has been proved and still remains an assumption from anatomy." They also state, "We are rather of the belief that the gall-bladder acts as a buffer or pressure-valve to relieve acute distention and protect the pancreatic tissue." This is the true function of a reservoir; and C. H. Mayo[18] states that the gall-bladder can enlarge so as to hold 200 c.c. while its normal capacity is 50 c.c. But by its power of concentration it becomes a great potential reservoir. Digestion is active for ten hours and quiescent for fourteen hours out of the twenty-four.[28] During these hours the bile is stored by condensation mainly and by bulk to some extent.

(3) Further, these authors contend that there is nothing specific about the action of magnesium sulphate solution, and state that "a more abundant flow of the 'B' bile is obtained by the injection of from 5 to 50 c.c. of a one-half per cent. solution of hydrochloric acid"; and, again, "with the use of any of a number of substances the characteristic 'A,' 'B' and 'C' biles, and mainly the so-called 'B' bile, is obtained quite as readily as with a solution of magnesium sulphate." They secured the same results with fibrinated and defibrinated pig's blood. These statements are completely contradictory to that of Einhorn[29] that the deepening of the color of the "B" bile is due to the "sulphate part of the salt (magnesium or sodium)." It is then suggested that the dark color of the gall-bladder bile is due to oxidation of its constituents. There is no proof for this; and there need be none, since a complete explanation is found in the condensation of the bile that occurs.

(4) Bassler, Luckett and Lutz further contend that the darker or "B" bile is obtained from the majority of cholecystectomized persons, and also from those very soon after the operation, before the biliary passages have had time to dilate. This opinion is quite divergent from

those of other workers upon this subject, and one feels that there is urgent need for further study of this aspect of the case.

Frank Smithies[30] writes thus: "In view of its quantity, and from the observation that after cholecystectomy this sudden gush of a dark-colored bile is not secured, it is evident that this material comes from the gall-bladder." This statement could be corroborated by quotations from others, but one from McCaskey[31] will suffice. "Taking into account all the available evidence, together with my own observations, I have no hesitation in affirming that the dark-colored 'B' bile, obtained by the Meltzer-Lyon method, is usually obtained from the gall-bladder." He then goes on to state that in about 70 per cent. of his cases the dark-colored "B" bile was obtained. In the remaining 30 per cent. it rarely ever happened that the light yellow bile underwent no changes, excepting in those cases in which the gall-bladder had been removed or there was good reason to suspect that it was not functioning because of obstructed ducts or other causes. The more gradual changes of color are due to a slower action on the part of the gall-bladder permitting a considerable amount of liver bile to mix with its escaping contents.

In the event of obtaining the "B" type of bile from persons after the removal of the gall-bladder one must bear in mind that in post-cholecystectomy cases there is a disposition in the biliary tubes to dilate. This may account for bile stasis and its assuming a dark color. In instances where this occurs very soon after the removal of the gall-bladder, there may have been dilatation prior to the operation as the result of obstruction, or the bile may be dark from admixture with blood, or the anæsthetic may have stimulated the liver to throw out more pigment, and also the liver may have been in an abnormal condition in connection with the morbid processes demanding the cholecystectomy.

In the statement of Bassler and his associates that "There is practically as large a portion of people having gall-bladders in which no stimulant causes the 'B' bile to occur," the "as large a portion" refers to persons previously mentioned who had no gall-bladders and among whom "here and there one is met with in which it ('B' bile) is not obtained with any stimulant." These very rare cases of persons with gall-bladders who do not yield the characteristic "B" bile belong to the class mentioned by McCaskey just quoted, who contends that, though the employment of the duodenal tube has its difficulties, "the results are worth while, and supply information of the highest importance that cannot be obtained in any other way."

Einhorn[32] bases one of his chief objections on the discovery that the changes of color from light to dark and from dark to light bile are not sudden but gradual. He concludes that if Lyon is correct, as soon as the common duct becomes emptied, the dark gall-bladder bile would appear, and that, when this supply became exhausted, there would be again a sudden change to the light-colored liver bile. In his observations these changes are not abrupt. But there are a number of possible reasons for this. In the first place unhealthy gall-bladders may respond more slowly and empty their contents over a longer period of time, during which some light-colored liver bile becomes incorporated. In the second place a normal duodenum, common duct, and gall-bladder need not necessarily respond with equal promptness, causing, thereby, the changes in color to be somewhat gradual in some of them. In the third place, the influence of a general anæsthetic must not be overlooked; for it is well known that sensation, motor power, and reflex activity are all greatly modified and often abolished in this condition. While the changes in color may be rapid, all now admit that they need not be so.

Next he noted that the dark-colored bile followed the instillation into the duodenum of a solution of sodium sulphate which does not relax the duodenum as does the magnesium sulphate solution. This had not been the author's experience. He has found that a solution of sodium sulphate does stimulate the flow of fluids from the mucous membranes of the duodenum and common bile duct, and incites a flow of the bile contained in the common duct. The bile in the gall-bladder then enters the cystic and common ducts and finds its way into the duodenum. This can be proven by studying what takes place when an animal is operated upon under local anæsthesia. It is not, therefore, necessary to regard the dark bile as due to the sulphate part of these salts. Crohn *et al.* found that magnesium sulphate solution had no influence on the color of the bile. With this statement we agree from observations made on bile collected from the hepatic duct, after injection into the duodenum of both magnesium and sodium sulphate solutions. In eleven cases of complete cystic duct obstruction, Whipple[33], after lavage of the duodenum by magnesium sulphate solution. recovered only light yellow-colored liver bile.

Einhorn further states, "I have also tried to study the effect of magnesium sulphate upon patients whose gall-bladders have been removed. In some of them I could observe a similar color play after the magnesium sulphate instillation." Here, however, there is always the possibility that in the "some," where the dark bile was obtained, there was dilatation of the biliary tubes to take the place of the absent gall-bladder. On the matter of color Whipple states "if, after magnesium sulphate instillation, there occurs a rapid flow of thick, dark green bile, the cystic duct may be considered patent and the walls of the gall-bladder contractile." In the six cases of Luckett [27] dark bile was always obtained by compressing the gall-bladder. This goes to prove the true location of the dark bile. The theories regarding the chemicals that cause it to reach the duodenum is another question.

He claims that sodium sulphate solution will cause the changes in the bile, although this salt does not relax the sphincter of Oddi and induce expulsion of bile from the gall-bladder. He contends, therefore, that the color changes are produced by the action of the magnesium or sodium sulphate on the liver; and suggests that it is the sulphate element that is the effective agent, as no change in color occurs after the use of sodium phosphate or bicarbonate. He also shows that magnesium citrate in a 10 to 13 per cent. solution does not alter the color of the bile or does so to a very slight degree. When the magnesium sulphate is employed in a high degree of concentration the more intense is the color at its acme. From these facts he concludes that the changes in color are due to the action of the sulphate salts on the liver and not by the opening up of the gall-bladder. He has also found similar color changes after the instillation of magnesium sulphate solution in cases where the gall-bladder had been removed.

He further goes on to state that the increase in the dark color follows a certain curve, gradually increasing until it reaches its acme, then slowly decreasing until there is a return to the usual yellow color or a lighter yellow. The magnesium sulphate was employed in cases of liver derangement associated with myocarditis, hypertrophic cirrhosis, and cardionephritis, and no change in color occurred. It would appear from these tests that the liver must be capable of functioning in a nearly normal way in order that the change in color may be manifested.

Einhorn states [33a] that the stronger the magnesium sulphate solution the darker the bile and the higher the specific gravity, and uses this as an argument in favor of the view that the solution has acted upon the liver rather than by evacuating the gall-bladder. But it may be explained on the ground that the strong solution empties the gall-bladder more rapidly, fully, and with less addition of liver bile. In this way the dark and concentrated bladder bile is recovered, accounting for the deep color and high specific gravity attributed to liver secretion. Further, he states that "After applying the magnesium test, an immediate repetition of the test frequently provokes a reiteration of the same reaction with its entire series of color plays." If the color was due to liver action this should always occur and not "frequently." The phenomena cited can be explained on the easy assumption that the gall-bladder did not fully empty itself the first time. This is known to happen, and it is common practice to follow up the first injection with a second one. Of the thirty-seven tests (Sept. 7, 1921) only four gave a clear bile all the way through the series of aspirations, whereas thirty-three gave a turbid bile at some stage of the aspirations. This would lead to the conclusion that the gall-bladder and biliary ducts were in a morbid condition, as otherwise the liver would not so frequently yield a turbid bile in response to the absorbed saline, and that it was from these morbid sources that the dark bile came. Much weight is attached to a case where the test yielded dark bile, and at the time of operation the cystic duct was completely blocked by a calculus. Here it was held that the dark bile came from the liver as the result of the absorption of the solution. But, again, it must be remembered that no bile could enter the gall-bladder, and consequently the reservoir of the biliary system, with its concentration function, was absent. The biliary ducts under these conditions dilate in order that they perform the dual function of holders and transmitters. His statements may be summarized as follows:

In the fasting state the bile in the duodenum is normally golden yellow and clear, but in pathological conditions of the biliary apparatus it is turbid, greenish yellow or dark brown and contains mucus, pus, and frequently some form of organisms, with cholesterin and bilirubin calcium crystals. In cholecystitis without stones the bile is turbid with mucus and pus. In cholecystitis with stones there are in addition cholesterin and bilirubin calcium crystals. In complete common duct obstruction no bile or pancreatic fluid is found in the duodenum, even after repeated lavage and aspirations. The duodenal bucket and string are helpful aids in differential diagnosis. Clear bile obtained by the tube or bucket and blood on the string point to a normal condition of the biliary organs and the presence of an ulcer. Turbid bile and no blood stain points to gall-bladder disease; while both turbid bile and blood stain indicate disease of the gall-bladder and ulcer.

In this statement it is made clear the cholecystitis gives rise to a turbid and dark bile, containing crystals and bacteria. When such bile reaches the duodenum, and is recovered through the tube, one has the typical "B" bile described by B. B. V. Lyon.

Turning to the views of Crohn, Reiss and Radin [34] it will be noticed that their objections to the teachings of Lyon may be summarized thus:

(1) They do not find a distinct muscular ring around the mouth of the common bile duct, but they do find scattered muscular fibres, and that there is a sphincter action, as claimed by Oddi [35], Hendrickson [36], Mann [37], and Herring and Simpson.[38] They found that gently touching the papilla of Vater with the finger or the application of faradic stimulation or chemical irritants promptly closed the duct. In from ten to thirty seconds relaxation occurred and the bile began to flow. They state that "cholagogues and solutions of salines and peptones had a tendency to encourage this flow of liver bile; but found no agent, applied in the duodenum, that caused the expulsion of the gall-bladder contents."

(2) In experiments upon dogs they did not observe any contraction of the gall-bladder after the placing in the duodenum of magnesium sulphate solution. As against this we have the positive observations of Sachs [39] who saw the gall-bladder collapse by emptying itself while the patient was undergoing an operation, an instillation of magnesium sulphate solution having been given. On this point Meltzer [40] states: "But the bile in the gall-bladder, as well as in the biliary ducts, is under a certain pressure which is sufficient to drive it into the intestines if the papilla of Vater is relaxed."

(3) They have doubts as to the dark or "B" bile coming from the gall-bladder after injection of the magnesium sulphate. In their own cases, however, they obtained this type of bile in 65 per cent. of their cases, and of these 62 per cent. revealed distinct pathological conditions at operation. In some of their cases where a dark bile could not be secured, complete obstruction of the cystic duct was found to exist. In these cases the magnesium sulphate did not give a dark bile by acting upon the liver and stimulating the excretion of bile salts and coloring matter, as one would expect from the studies of Einhorn [32] and Dunn and Connell.[26] They injected into the gall-bladder a solution of methylene-blue in normal saline, and on opening the duodenum and stimulating it,

only clear liver bile appeared. While they did not find any substance that caused the expulsion of bile from the gall-bladder, they state that cholagogues, solutions of peptones and salines did increase the flow of liver bile.

(4) They then take up the question of the color of the bile, and conclude that the dark bile, or "B" bile of Lyon, is not from the gall-bladder, but from the biliary tubes. They found that the magnesium sulphate solution did increase the flow of bile, but had no influence on its color. In some cases that went to operation and were found to have diseased conditions of the gall-bladder, no dark "B" bile was obtained by the duodenal tube. In one case dark bile was secured though the gall-bladder had been previously removed. They also found dark-colored bile in cases where the gall-bladder was found to be normal. Further, they found that bile aspirated from gall-bladders at the time of operation was nearly always darker, a deep dark brown shade, and more mucoid in consistency than the "B" bile in the duodenum. Microscopically, no similarity could be found in the sediments of the two compared fluids.

(5) Duodenal lavage was tried in a few cases of cholelithiasis, where chronic cholecystitis was suspected to be present, without change in the clinical course. In cases of incomplete obstructive jaundice due to stone or new growths, a freer flow of bile took place after the lavage than before it. In catarrhal jaundice the icterus disappeared within a reasonable time. Whether sooner than it would have done otherwise it is difficult to judge. It will be noticed that if the results of those investigators are correct, those obtained by Lyon must be wrong; for the latter states very definitely that magnesium sulphate solution does relax the Oddi muscle and, at the same time, contracts the gall-bladder muscle. All discussion about the color of the bile is of much less importance than is this physiological problem.

Here are divergences of opinion that call for some comment. Einhorn [29] states that "the high concentration of the salts introduced may have something to do with the increased outflow of bile and other juices." He contends that a solution of magnesium sulphate does not empty the gall-bladder into the duodenum. Crohn, Reiss and Radin, already quoted, agree that magnesium sulphate solution does not induce contraction of the gall-bladder, but differ from Einhorn in declaring that it does not even change the color of the bile. Drs. C. F. Tenney and H. S. Patterson [41], after a careful study of a case with biliary fistula where no bile entered the duodenum, found that the amount of bile was not increased by the injection of magnesium sulphate solution. They also make it quite clear that the solution has no effect in darkening the color of the bile. In this they differ from Einhorn. They state that normal bile introduced into the duodenum does increase the flow of liver bile.

Endorsement of Non-surgical Duodeno-biliary Drainage.—The teachings of Lyon are well known and may be read in a recent review of the subject in answer to a number of his critics.[40] The results attained and the opinions advanced by a few of those who have investigated this method of biliary drainage and found it both a valuable aid in diagnosis and treatment are set forth in the following summary:

Hopkins[42] reports some cases of acute catarrhal jaundice in the treatment of which he found much assistance from the use of the duodenal tube and the injection into the duodenum of 50 c.c. of a 25 per cent. solution of magnesium sulphate. The treatment consisted in passing the tube in the fasting state, and the stomach aspirated. Calomel, grains 1½ (0.095 gram), was given, and the duodenal contents aspirated over a period of two hours, when 50 c.c. of fluid were recovered. The day following the calomel was again given, and the magnesium sulphate solution injected, after which 250 c.c. of fluid were obtained by aspiration in two hours. This result was secured in several patients, and the conclusion arrived at that while the calomel induced a copious flow of bile, the magnesium sulphate solution brought it promptly down into the duodenum by the relaxation of the outlet of the common bile duct. The improvement of the patients under this method of treatment was most gratifying and rapid. This author remarks that any disagreeable features connected with the use of the tube is more than counterbalanced by the relief from the unpleasant symptoms of catarrhal jaundice. "The relaxing of the duct by the magnesium sulphate seems to be a useful auxiliary measure in attaining the rapid results."

White[16] discusses the question of the drainage of the bile passages and gall-bladder. He states that it is admitted on all hands that after the injection into the duodenum of the magnesium sulphate solution there is a flow of dark bile, but some hold that this is due to the emptying of the gall-bladder, while others contend that it results from the solution stimulating the liver to excrete a greater amount of coloring matter. White states that the important point is that the biliary passages are drained. He expresses the opinion that the gall-bladder may not always be wholly emptied, and that the emptying may vary greatly in cases both as to rapidity and completeness, and that more than one stimulation may be required. This may lessen the value of the method for diagnosis and treatment.

For the purpose of microscopic examination it is important that the mouth and stomach should be washed clean to prevent contamination of the duodenal fluid. In spite of care there may be various forms of cells from the nasopharynx find their way into the aspirated fluid; and some of the acid contents of the stomach may mix with the bile causing some turbidity by precipitating the bile salts. In true biliary sediment there is usually deep bile-staining of the cells. There are present leukocytes, cuboidal duodenal cells, and columnar epithelium from the ducts and gall-bladder. In cases of disease these features are marked, and there are present pus cells, and perhaps blood corpuscles, and mucus. In normal conditions these findings are scant. In all biles amorphous forms of cholesterol and bile salts are present. When abundant, or a gritty sediment is found, there is a probability of gall-stones. Cultures have revealed the presence of colon bacillus, staphylococcus aureus, streptococcus, bacillus proteus and subtilis, and staphylococcus albus. About one-fifth of the cultures were negative. Acute and subacute cholecystitis yielded most frequently positive cultures, while chronic cholecystitis and gall-stone cases gave most frequently sterile cultures. In these cases the bile is darker than normal, thicker, contains mucus, and much cell elements. Chronic cholecystitis and some gall-stone cases give a clear bile of increased viscosity and sterile cultures as the usual condition. Absence of the dark "B" bile, but a flow of yellow bile indicates obstruction of the cystic duct, as was verified several times at operation. Absence of bile and presence of pancreatic ferments after several careful aspirations indicates obstruction in the common duct.

E. C. Cutler in discussing White's paper said that in cases where he did not find the dark bile, but a continuous flow of the lighter bile, he diagnosed obstruction of the cystic duct. There were about 18 cases in which this diagnosis had been made, and at operation this was found to be incorrect only once.

Pope[43] records his impressions after an experience of more than 1,500 single drainages, and states: "It is to my mind the first and so far the only method of really studying the biliary tract." He finds:

(1) It is useful where there is a "mild or low-grade infection of chronic type, with little or no distinctive gall-bladder symptoms, but often many gastric, intestinal and nervous symptoms," and

(2) In gall-bladder infections resulting from disease of the colon, from which bacteria penetrate to the deeper layers of the colon wall and are carried by the leukocytes to the portal veins and lymphatics, and thence to the liver. A vicious circle is established by the colon infecting the biliary passages in this way, and these again infecting the intestinal canal.

(3) Some cases do not seem to be disturbed by the use of the tube, and feel quite well afterwards. Others feel weak, tired and listless, and are benefited by hot broth in small amounts and no solid food for some hours. As a general rule food should not be taken for several hours after a drainage. They may have a restless night unless properly medicated. Sometimes large quantities of bile are passed per annum. Following the drainage of septic bile, there may be "arthritic, myositic, fibrositic and neurotic aches and pains from absorption of toxic products." In such cases enough of Jutte's solution should be given after the drainage to clean the tract. In cases of absorption of toxic matter from the biliary passages, drainage clears the skin, ameliorates the headaches, and improves the health.

(4) The author has not found drainage of much help in constipation. Such cases call for treatment in addition to the biliary drainage; but, if the indications are such as to require drainage, it is of assistance to the other measures adopted for the relief of the patient. "A patient with infection in the great glandular, saccular and tubular systems of the liver is sick, not with a purely local lesion to be drained, but sick all over, toxic, and to trust solely to drainage and drainage alone for relief, is but to court failure and disaster."

(5) Several varieties of parasites have been discovered after drainage in the duodenal contents. The most commonly found parasites are forms of the hookworm (anchylostoma, necator and uncinaria Americana); but strongyloides intestinales, endamœba histolytica, amœba coli, amœba dysenteriæ, flagellatæ, and trichomonas intestinales are also found, though less frequently. This shows one of the valuable facts to be obtained by this method of diagnosis.

(6) All cases should be supervised personally by the physician himself. Thick, foul bile may be free from pus, while thin, clear bile may be full of pus and crystals. Laboratory study of each and every biliary drainage is essential. What should next be done must be determined by the result of this investigation. In some cases it may be well to make use of the duodenal bucket to secure the contents of the viscus.

(7) In some patients it is very difficult to secure passage of the bulb of the tube or the bucket through the pylorus owing to spasm. Hot water, benzyl benzoate, and atropin hypodermically may fail, and, for such cases, this author recommends pressure on the back on each side of the fifth dorsal vertebra. After the administration of the magnesium sulphate solution, concussion over the first, second and third lumbar spines aids in relaxing Oddi's muscle.

(8) He prefers a right-side semi-abdominal position for good drainage. In a few cases the erect or left-side position will yield better drainage.

McClymonds[44] in dealing with this method of diagnosis remarks: "When the duodenum is properly douched with magnesium sulphate solution, failure to obtain gall-bladder bile while securing liver bile means definite pathology, and with the exception of external pressure from glands or growths, means disease of the gall-bladder or cystic duct. This may be obstruction due to inflammation of the mucosa and swelling, plugging with mucus, the impaction of stone, kinks, stricture, malignant growths, the gall-bladder may be atrophied, entirely filled with stones or sand, the contents too viscid to flow, or the musculature too weak to bring about the expulsion of its contents. A rapid flow of 'B' bile indicates a normal gall-badder, while a slow or interrupted flow, requiring a second or even third stimulation, points to a morbid state of the viscus, or constriction or partial obstruction of the duct."

He agrees with Lyon that the appearance, the cytology and the bacteriology of the "A," "B" and "C" biles give information of great value as to the location of disease in the biliary tubes and gall-bladder.

This author had performed seventy drainages on forty-two cases. In two no bile could be secured and at operation the common duct was found obstructed by calculi. In another case liver bile, but no gall-bladder bile, was obtained. In this case operation revealed a stone in the cystic duct. In a number of the cases the quantity of "B" bile was much in excess of normal, indicating stasis of the bladder. It is in such cases that the treatment by drainage proves so valuable. The gastric symptoms and headaches are relieved, the skin clears, the health improves, and the weight increases. He concludes with these words: "We believe that the method will prove of the greatest aid in the clearing up of many obscure abdominal conditions, enabling us to make a diagnosis at a much earlier stage in the pathological process, as well as giving us the best method of treatment in many gall-bladder and hepatic diseases."

Johnson [28], in a very exhaustive study of the functions of the gall-bladder and the Lyon method of biliary drainage, made a number of important observations.

(1) Repeated tests on dogs showed that the sphincter muscle of the common bile duct relaxes under the influence of 15 c.c. of a 25 per cent. solution of magnesium sulphate.

(2) Tests made on dogs that had fasted for sixteen hours showed definitely that the injection of 15 c.c. of a 25 per cent. magnesium sulphate solution produced the three different phases of bile described by Lyon.

(3) In two dogs the abdomen was opened under local anæsthesia, and the duodenum injected with saline solution. There was observed a slight collapse of the fundus of the gall-bladder followed by a minute wave of contraction which ended by producing a fullness about the opening of the cystic duct which was succeeded by a fullness of the duct itself. During digestion it has been found to contract in a weak and irregular manner about every five minutes.

(4) In one of the dogs the tube was placed in proper position. The abdomen was then opened and 15 c.c. of a 25 per cent. solution of magnesium sulphate were injected. The peristaltic contractions of the duodenum became less active. The changes in the color of the bile to a certain extent could be seen.

(5) In one of the dogs the gall-bladder was removed, and the test showed the absence of the dark-stage bile or "B" bile.

(6) If the test is applied in fifty to sixty minutes after it had been used, the gall-bladder phase is obtained, showing that the viscus had not fully emptied itself. It is emptied mainly by the movements of adjoining organs and suction by the duodenum.

(7) The author raises the question that, as in about 85 per cent. of infections of the gall-bladder the infection is carried by the blood stream [45] and the walls of the viscus primarily attacked, drainage of the biliary tubes would not effect relief.

(8) After controls had been secured repeated tests in a cholecystectomized dog showed the absence of the gall-bladder phase. This indicates that the magnesium sulphate solution does not darken the liver bile.

Whipple [33] has contributed a report on twenty-seven cases. Of these twenty-four came to operation. In two of these the characteristic "B" bile was found, and in both cases there was cholecystitis. In three cases no bile at all was obtained, and in these the common duct was obstructed. In eleven cases bile of the "A" and "C" types were secured, but no bile of the "B" type. In each case the cystic duct was closed. In eight cases "B" bile was not found, though no special disease of sufficient degree was discovered to account for the cystic bile not appearing in the duodenum. Whipple regards his series of cases as confirming the value of the Lyon method of draining the biliary channels.

Friedenwald and Morrison[46] as the outcome of their studies lay down a number of definite statements: (*a*) Failure to secure gall-bladder bile means obstruction of the cystic duct; (*b*) failure to secure any kind of bile means obstruction of the common duct; (*c*) dark turbid bile with numerous microörganisms, quantities of mucus and pus, and an increase in cell elements, indicate an infected gall-bladder; add to this pain and the presumption is strongly in favor of the presence of calculi; (*d*) in one hundred and forty-nine cases of chronic cholecystitis brilliant results were achieved by biliary drainage, and many organisms were discovered; (*e*) in one hundred and twenty-three cases of biliary stasis they found that daily treatment by drainage extending over several weeks resulted in great benefit, this condition being due to atony of the gall-bladder, however when the "B" bile exceeds 90 c.c. there is stasis; (*f*) this method of examination is of particular value as it enables us to diagnose the conditions of the gall-bladder and biliary tubes at any early stages of disease.

Simon[47] in an experience with thirty cases of disease of the gall-bladder and bile ducts found this form of drainage to yield excellent results, especially in catarrhal jaundice. The pain, when present, disappeared, the jaundice lessened, the fever abated, toxic conditions became less pronounced, and health improved.

Smithies[15] expresses his views in these words: "All who have had actual experience with the Meltzer method agree that, by this method, one is able to recognize disease of the gall-tract earlier than by any other clinical procedure. Since this is true, then by carful regimen, it would seem that we have an opportunity to directly treat liver and bile passage affections before they become sufficiently advanced to produce malformations, obstructions, calculi or malignancy."

In the article just quoted it is stated that 650 biliary tract drainages had been performed on 300 patients. It is also stated that on patients who had undergone surgical operations, but were still afflicted with dyspepsia, this form of drainage revealed the fact that "70 per cent. of them had inflammation with infection still persisting in the large or small bile ducts." He further states that: "As a result of our experience, we venture the opinion that, except in rare instances (acute obstruction, perforation, marked inanition, etc.), operative procedures upon the gall-tract are not justified without previous diagnostic biliary tract aspiration. Such aspiration, properly performed, permits the surgeon to prognose in a striking fashion, before laparotomy, the condition which operation will disclose."

It is also pointed out that in a study of 1,000 operative cases the bile was found to yield, on culture, bacteria in 63 per cent. In very chronic cases where surgical intervention became warranted no microörganisms may be found. In these cases showing no infection the microscope yields other evidences of disease, such as more than 100 c.c. gall-bladder bile, abnormal appearances of the bile, finding small calculi, pus, excess cholesterin, bile salts, mucus, gritty deposit, and many and varied cells. By this method of diagnosis, disease of the biliary apparatus is sometimes discovered where the patients complained of feeling ill and revealing only toxic symptoms. From his experience he attaches much value to it as a diagnostic and therapeutic measure. In the discussion of this subject by Smithies, Karshner and Oleson[48] there is a very complete review of the advantages of this method of gall-bladder drainage, and an examination of the objections that have been raised against it. They accept the teachings of Doyon, Meltzer and Auer regarding the existence of Oddi's muscle and its function. They state that magnesium sulphate is a very suitable agent for the purpose of relaxing this muscle and also the muscle fibres of the duodenum. They contend that, while this salt may act on Oddi's muscle, when taken by the mouth, it should be administered through the duodenal tube to secure constant results. It is also stated that disease of the gall-bladder interferes with its contractile power and explains why some operators think it devoid of such power. If the patient is not deeply anæsthetized one can see dilatation of the duodenum with contraction of the gall-bladder and bile ducts, and an out-pouring of bile. These writers state that the dark color of the "B" bile is not due to the magnesium sulphate. In this they differ from Einhorn and Dunn and Donnell. They ask the question: "Where, in the course of the bile passages from the papilla of Vater to the liver, can such accumulations of a special type of bile (chemically, cytologically, and qualitatively) be held, if not in the gall-bladder?" They answer their own query by stating that after cholecystectomy dark bile may come from the ducts (presumably because these dilate).

The experience covered over 1,500 drainages. Of this number 679 are tabulated as follows:

Specimens Secured	*Number*	*Per cent.*
After one instillation 15% $MgSO_4$ Solution	461	67.9
After two instillations 15% $MgSO_4$ Solution	106	15.6
After three instillations 15% $MgSO_4$ Solution	17	2.5

Specimens Not Secured	*Number*	*Per cent.*
Bulb did not leave esophagus	1	0.15
Bulb did not leave stomach	35	5.15
Tube knotted	2	0.29
Tube withdrawn on account of neck pain	1	0.15
Tube constantly vomited	1	0.15
Tube vomited (osteomalacia case)	1	0.15
Tube imperfect	1	0.15
Profuse duodenal haemorrhage	1	0.15
Only blood-stained fluid secured	1	0.15
Bulb in jejunum	3	0.45
One instillation $MgSO_4$ solution failed	18	2.65
Two instillations $MgSO_4$ solution failed	25	3.68
Three instillations $MgSO_4$ solution failed	4	0.58

According to this table only 47 or 7 per cent. failed, though the solution had reached the duodenum. Of these 18 had been stimulated only once, and 25 only twice.

Failure to obtain gall-bladder bile, notwithstanding that the end of the tube is in the duodenum, may be due to absence of the gall-bladder, to cystic duct obstruction by calculus pressure or growth, to gall-bladder atony, to very thick gall-bladder contents, tumor of the viscus or ducts, or to adhesions. As shown by Johnson,[33] if the end of the tube is too high or too low in the duodenum bile may not be secured. Practical segmental biliary tract drainage in persons without biliary symptoms can be secured in 95 per cent. of cases. Departures from the "A," "B" and "C" sequence commonly indicate disease of the gall-tract and liver.

The time required to effect proper drainage is shown by the following table:

Less than two hours	1
Two to four hours	67
Four to six hours	291
Longer than six hours	188
Total	547

Value in Treatment.—Much of what might be said under the caption of treatment has been touched upon in the several sections already covered. In the present section a brief resumé only will be given.

Smithies, Karshner and Oleson [40] lay down a lengthy series of conditions in the treatment of which duodenal biliary drainage has been found useful.

(1) Acute choledochitis, cholecystitis or hepatitis in association with acute infectious ailments, as pneumonia, etc., or arising as a limited biliary tract disease.

(2) Hepatitis with faulty duct action from ptomain, lead or phosphorus poisoning, and acute yellow atrophy, such as may follow intravenous or muscular injection of arsenical preparations.

(3) Biliary stasis in association with acute or chronic heart trouble, or with cirrhosis of the liver, or with infection in anemias such as hemolytic, pernicious, Banti's syndrome, or with stasis or infection during diabetes, pancreatitis, or exophthalmic goiter where operative interference is not possible or can be performed to but a limited extent.

(4) Attacks of biliousness in conjunction with migraine, epilepsy, etc.

(5) In rheumatoid infections after the foci of infection have been removed but the biliary tract involvement and the rheumatoid condition continue.

(6) In cases of pyloric or duodenal ulcer recurring with bilious attacks, or atypical ulcer manifestations, and when reliable surgery is not available or the subject is not suited.

(7) In instances of intestinal stasis with biliary and liver types of dyspepsia, and attacks of mucous colitis.

(8) When after operation on the biliary organs derangement of function continues and further surgery is not possible.

(9) In cases of infection of the biliary organs without gross mechanical defects, stones, or new formations.

Stockton[49] found injections into the gall-bladder, through an external opening, of magnesium sulphate solution valuable in a case of severe liver and duodenal infection.

Niles[50] regards the employment of this method of draining the biliary passage as worth using in jaundice, cholecystitis, choledochitis, migraine, and biliousness. Many cases may in this way be cured, and never become surgical subjects.

Simon[47] found the injection of magnesium sulphate solution into the duodenum brings prompt relief in a number of cases, the pain, local symptoms, and toxic features disappearing.

Pope[43] expresses himself as of the opinion that there is merit in this method of drainage and is "a helpful diagnostic and therapeutic measure"; and also "the Lyon-Meltzer method offers a new and promising method for the diagnosis and drainage of the biliary tract"; and "biliary and colonic infections are practically always present in the same person."

McClymonds[44] reports a case where, after surgical drainage of the gall-bladder, and later on its removal, painful attacks were of frequent occurrence. The Lyon method of drainage gave great relief. He then goes on to say: "We believe that the method will prove of the greatest aid in the clearing up of many obscure abdominal conditions, as well as giving us the best method of treatment in many gall-bladder and hepatic diseases."

White[16] puts his views thus: "I believe that the method frequently relieves biliary stasis, will help to clear out infection, will take a load off the liver, and, if successful, will frequently prevent more serious damage to the liver, gall-bladder and bile passages." He suggests that it may be useful where there is a question as to the immediate need for surgery, and also in cases where surgery has effected only a partial cure. "By relieving biliary stasis it does much to prevent catarrh, infections and stones. The best field for its use is in the milder and moderate grade of cholecystitis and choledochitis cases, where no marked obstruction exists. Many such cases are greatly relieved, or apparently cured, following this treatment."

Einhorn[32] states the case as follows: "These salts (magnesium and sodium sulphate) seem to stimulate this organ. The aperient waters are ordinarily taken by mouth. Would they not manifest quicker and better action when instilled directly into the duodenum?"

Friedenwald and Morrison[46] conclude their article thus: "While this method of treatment is still in its infancy and is as yet too new to draw conclusions regarding its end results, there can be no question that it furnishes us with a means of affording definite relief in certain biliary affections for which hitherto radical measures have been required. It is an extremely valuable aid in the treatment of catarrhal jaundice, biliary stasis, and gall-bladder infections, and is helpful in relieving the infection which may still remain following gall-bladder operations."

Lyon, Bartle and Ellison[11] have this to say: "As a result of four years of practical application of this method of diagnosis and treatment of biliary-tract disease in a series of over 400 cases, we can earnestly express our belief that it is now a proved and rational method in the successful treatment of early cases, and is possessed of great possibilities of future development in the prevention and control of many of the later and more severe states of disease of the biliary tract, and we trust it may hopefully be extended to certain diseases of the liver, of the pancreas and of some of the more serious of the problem diseases of internal medicine.''

"In the field of treatment it is certainly the method of choice for biliary stasis, gall-bladder atony, and in the early states of catarrh and infection. It certainly is useful as a post-surgical 'follow-up' plan of treatment in many cases (Lyon[51]).''

Synnott[52] adopts Lyon's technic both in the matter of diagnosis and treatment, and is a firm advocate of this method of draining the biliary passages, as shown by these words: "To establish early diagnosis and treatment of biliary stasis is most important because stasis with its concentrated bile, and precipitation of its crystalline chemical ingredients, plus catarrh, plus infection, means gall-stones. The technic of Lyon enables us to do this with absolute positiveness and it is the only way in which a diagnosis can be made in every case.''

His treatment may be summarized as follows:

(1) While the metal tip is still in the stomach, the contents of the viscus are withdrawn for examination.

(2) The tip is then allowed to pass into the duodenum, and the magnesium sulphate solution is instilled. The three types of bile are then secured.

(3) The stomach and duodenum are lavaged with some of the disinfectants recommended for treatment, as the permanganate of potash.

(4) The duodenum is then slowly lavaged with 250 c.c. of Ringer's solution to which has been added sodium sulphate. This causes a thorough and painless movement of the bowels.

(5) This treatment is employed about once a week, and as the patient improves, less frequently.

(6) He reports cases where the results have been most gratifying.

These numerous expressions of a favorable character coming from many centers of medical teaching, and from the pens of highly trained and competent practitioners carry much weight. But, as has been said by several writers, sufficient time has not yet elapsed to enable the profession to lay down definite predictions regarding its value as a therapeutic measure. Failure or success is going to depend so much upon the skill with which the method is carried out. On this point the words of Lyon[47] should be borne in mind: "You will say this is highly specialized work and requires unusual specialistic knowledge. This is true. This is no field of diagnosis for the tyro.'' He then goes on to state that the house doctor, the nurse, and the laboratory worker must be thoroughly trained for the part each is expected to perform. What is true of diagnosis is equally so of the application of this method of gall-bladder drainage as a therapeutic measure.

It would appear from all the experiences so far that the earlier this treatment is instituted the better the prospects. In discussing its value Bassler *et al.*[21] remark: "There are many who do fairly well, but our belief to-day is that the less the pathology the more the benefit. Apparently there is benefit to be obtained in the simple catarrhal conditions mainly when this is in the ducts." This is from a severe critic of this method, but agrees largely with its advocates, who contend that one of its chief uses lies in the fact that it proves so valuable in atony of the gall-bladder, in cases of biliary infection, and in catarrhal states, whereby many of the serious results of these conditions are averted, and many cases cured that would otherwise go ultimately to the operating table. "The less the pathology the more the benefit" may be a eulogy rather than a commendation. Lyon[18] states "cholelithiasis remains entirely beyond the scope of this treatment." So also it may be said that when the gall-bladder and biliary ducts are distorted from chronic disease, but little can be expected from this form of gall-bladder drainage. Any form of treatment that will prevent the occurrence of these severe and advanced cases, demanding major surgical operations, should be welcomed by all, and herein lies the hope of the Lyon non-surgical duodenobiliary drainage.

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(Suppurative and Non-suppurative)

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Etiology.—*Acute Cholecystitis.*—The etiology of acute cholecystitis resolves itself into a question of some infection. The presence of calculi, some obstruction in the biliary ducts, or some foreign body, as a parasite, act only as predisposing causes. The direct or exciting cause has been set down as some toxin reaching the gall-bladder. There is little to support this view, and it is doubtful whether toxin is ever a cause. Injuries or traumas have also been given as an exciting cause. Here it may be remarked that an injury may only pave the way for some microörganism to act, the injury *per se* not actually inducing the cholecystitis. Let us consider the infections that may attack the gall-bladder and the route they may take. Some reach the gall-bladder through the hepatic artery, and in this way pneumonia, influenza and septicemia may cause an attack. Infection may also reach the gall-bladder through the portal circulation, and this is the likely route in typhoid and colon bacilli infection. It is doubtful if either of these organisms, or, indeed, any organism, finds its way to the gall-bladder along the common and cystic ducts.

A great variety of organisms have been found. The typhoid, paratyphoid and colon bacilli are recognized as causes. The varieties of streptococci and staphylococci are present in some cases. The pneumococci of several varieties give rise to some very acute forms of cholecystitis and may cause it primarily without involving the lungs. Not infrequently several bacteria, or a mixed infection, may be present. If the colon or intestines become adherent to the gall-bladder, the *Bacillus aërogenes capsulatus* and other organisms may pass directly into it.

Chronic Cholecystitis.—Chronic cholecystitis is generally the result of an acute attack. It may be the result of the irritation of calculi favoring infection. Any condition that interferes with the function of the gall-bladder—such as constipation, improper habits, tight lacing, or a sedentary life—may prove factors in the causation of chronic cholecystitis.

Symptomatology.—CLINICAL HISTORY.—(1) *Acute Catarrhal Infective Cholecystitis.*—In the mildest forms, this condition may be overlooked, and when it occurs in connection with other diseases, as typhoid fever, it may escape detection. The pain may be attributed to the original disease, and, if there is stupor, the patient may not complain of pain. The existence of adhesions shows that cholecystitis is not uncommon, and proves a cause for the formation of gall-stones. The pain varies in intensity, but is usually paroxysmal and resembles that of gall-stones; it is caused by spasm in the gall-bladder. If the serous coat is inflamed the pain is continuous, and may closely resemble appendicitis. There is tenderness over the gall-bladder, and especially at the tip of the ninth costal cartilage; and pain and hyperesthesia may be felt at the eighth and ninth dorsal segments. The muscles over the gall-bladder are rigid, and the breathing is frequently shallow and jerky. The gall-bladder may be found enlarged; it may move with respiration, and can be moved from side to side. When the cystic or common duct is inflamed or occluded from any cause, the distention of the gall-bladder is usually present. If there is peritonitis in the area of the gall-bladder there may be distention of the intestines, and the gall-bladder may not be palpable. In such cases there is likely to be vomiting. Jaundice is very rare, and the liver is not enlarged unless there is some complication or obstruction in the ducts. In mild cases there may be no rise of temperature, while in severer cases, especially with involvement of the tubes, or peritoneum, there is usually more or less fever.

(2) *Membranous Cholecystitis.*—This condition has been described under the names "croupous cholecystitis" and "fibrinous cholecystitis." The symptoms are similar to those met with when gall-stones are present. Membranous casts may be discovered in the stools, and the real condition in this way revealed. In some cases both gall-stones and membranous cholecystitis may be present at the same time. There is a greater liability to jaundice than in catarrhal infective cholecystitis, as the common duct may become occluded by the membrane cast off from the gall-bladder.

(3) *Suppurative Cholecystitis.*—The symptoms in this condition of the gall-bladder may vary a good deal, as they may be quite local to the organ or part of a more general inflammation involving the peritoneum. The patient complains of pain, and there is tenderness on pressure. The muscles in the region of the gall-bladder are rigid. The gall-bladder is sometimes distended and palpable. Due to adhesions, the shape and position of the gall-bladder may vary. It may be so contracted that it cannot be felt, or it may be obscured by distention of the intestines with gas or by the rigidity of the abdominal muscles. The pain is constant but may become very severe at times, like that of hepatic colic. The temperature is raised and is accompanied by rigors. The pulse is increased in frequency and may become very rapid—an indication for operation. The inflammation is spread to the peritoneum with the effect of interfering with intestinal peristalsis, causing distention and symp-

toms of obstruction. The inflammation may extend to the common or hepatic duct and give rise to jaundice. The spleen is sometimes enlarged and there is leukocytosis, ranging from 10,000 to 30,000. When the infection is the *Bacillus typhosus* the increase in the number of leukocytes is not marked. Albumin may be present in the urine, and infection may reach the kidneys and set up a pyuria. In severe cases with peritonitis the symptoms may closely simulate peritonitis arising from perforation of the intestines or the appendix.

(4) *Phlegmonous and Gangrenous Cholecystitis.*—The symptoms in these cases are acute and severe. There is severe pain in the region of the gall-bladder, and distinct tenderness on pressure. If the ducts are involved there may be jaundice, though this is not constant enough to become a symptom of value. Peritonitis speedily comes on in the region of the gall-bladder, and is liable to become general. Gangrenous cholecystitis is an advanced form of the phlegmonous cholecystitis. As the gall-bladder may ulcerate, slough or rupture, a spreading infective inflammation results, with grave toxemia. There may be marked pain and tenderness in the eighth and ninth dorsal segments. A moderate degree of leukocytosis is usually present.

(5) *Chronic Cholecystitis.*—The symptoms of this condition very closely resemble those of gall-stones. There are periods of pain, often colic, and periods of ease. In the intervals between attacks of pain the gall-bladder may be detected somewhat distended. In this condition, as in gall-stones, there may be pain and tenderness at the eighth and ninth dorsal segments. In cases of chronic cholecystitis there is less likelihood of jaundice than when the biliary colic is due to the presence of gall-stones. In some cases of gall-stones, however, the common duct may not be completely occluded and the jaundice may be moderate in degree and the pain no greater than when the result of cystitis. If gall-stones are recovered from the feces this would enable a differential diagnosis to be made between cholecystitis and cholelithiasis.

PHYSICAL FINDINGS.—(1) *In acute catarrhal infective cholecystitis* one of the first clinical findings is tenderness on pressure. This tenderness is located in the right hepatic region, but is most pronounced at the tip of the ninth costal cartilage. Sensitiveness of the skin to touch may be found in some cases at the eighth and ninth dorsal vertebræ. This sign is found in at least 75 per cent. of the cases. The upper portion of the right rectus and abdominal muscles are more or less rigid on palpation. Sometimes the gall-bladder may be felt or seen as an oblong-shaped swelling, as it is distended by exudates that cannot escape, owing to the inflammation affecting the cystic duct. There may be tympanitic distention of the intestines in the right hypochondrium, due to peritonitis. In mild cases the temperature is normal, but it may be raised if the serous coat or ducts are involved.

(2) *In membranous cholecystitis* the physical findings are similar to those noted in attacks of gall-stones. The discovery of membranous casts in the stools is distinctive of the condition.

(3) *Suppurative cholecystitis* is marked physically by tenderness on pressure and rigidity of the muscles in the right liver region. The abdominal reflex on the right side may be absent. The gall-bladder can be felt as a pear-shaped enlargement extending downward and inward from the tip of the ninth cartilage. If the gall-bladder communicates with either the colon or intestine it may be tympanitic. Cases of some duration may lead to enlargement of the liver downward. The gall-bladder may be so elongated as to be palpable in the right fossa and give rise to doubt as to the possibility of appendicitis. The temperature is raised and there may be rigors. The pulse is increased in frequency. The spleen is sometimes enlarged.

(4) *In phlegmonous and gangrenous cholecystitis* the physical signs are those of suppurative cholecystitis, but more severe and rapid in their progress, and with much severer general and local symptoms.

(5) *Chronic cholecystitis* may present an enlarged gall-bladder that is free from tenderness during the interval between attacks of pain. The skin may be tender over the eighth and ninth dorsal vertebræ. The physical signs, however, are very indefinite, and the diagnosis must be made on the general symptoms and history of the case.

LABORATORY FINDINGS.—These various forms of cholecystitis yield few laboratory tests. In some cases organisms may be absorbed from the gall-bladder and infect the urinary tract, and colon bacilli have in this way been detected. When jaundice occurs, bile coloring matter and bile acids may be found in the urine. In most cases where jaundice occurs it is only partial, and consequently the urine may contain urobilin. In suppurative, phlegmonous and gangrenous cases there is usually a leukocytosis varying from 10,000 to 40,000.

Diagnosis.—(1) *Acute catarrhal infective cholecystitis* with enlarged gall-bladder must be differentiated from the colon distended with feces, a displaced kidney, a tumor of the kidney, or a hydronephrosis. Cholecystitis with enlargement of the gall-bladder and paroxysms of pain may closely resemble hydronephrosis with painful crises. At first the enlarged gall-bladder is pear-shaped, and later becomes elongated. A floating kidney is more movable than the gall-bladder and less fluctuating, and if the gall-bladder is pushed backward it returns when the pressure is removed, whereas a floating kidney remains where placed so long as the patient is recumbent. A floating kidney tends to slip away from the hand while the gall-bladder does not. A pyloric tumor is transverse to the axis of the gall, and is hard and tender. Tumor of the colon is not so well outlined as an enlarged gall-bladder, and is usually transverse to it. A fecal mass can generally be determined by the presence of fecal accumulations elsewhere, and their removal by an enema. In biliary colic the pain is more severe than in cholecystitis, while the tenderness, intestinal distention and enlargement of the gall-bladder are more in evidence in cholecystitis. Great care must be taken to distinguish cholecystitis from appendicitis and pyelonephritis. In the case of cholecystitis the tumor can be traced from above downward, while in that of appendicitis it may be traced from below upward. Pus in the urine may clear up doubt between cholecystitis and pyelonephritis.

(2) *Membranous cholecystitis* must be diagnosed by the finding of casts and a history that closely resembles that of gall-stones, with which it is usually associated.

(3) *Suppurative cholecystitis* may resemble subphrenic abscess, duodenal ulcer, or appendicitis in an abnormally located appendix. In the case of cholecystitis the tumor moves with respiration; duodenal ulcer gives the history of pain a few hours after taking food, and sometimes of blood in the stools. A perforated duodenal ulcer and suppurative cholecystitis may be indistinguishable. From appendicitis it must be differentiated along the lines already laid down under catarrhal cholecystitis. The resemblance of suppurative cholecystitis to pyelonephritis, pneumonia or pleurisy on the right side may be close. If the gall-bladder perforates and sets up a general peritonitis this latter must be distinguished from the peritonitis caused by a ruptured appendix or a perforated gastric or duodenal ulcer. The material found on opening the abdomen will clear up the doubt, and the history of the case in its early stage should be carefully inquired into for evidences of one or the other of these conditions.

(4) *Phlegmonous and gangrenous cholecystitis* usually has a history of former attacks of cholecystitis. When there is a localized inflammation in the upper right quadrant the differential diagnosis rests upon the points already mentioned. When the case has assumed the characteristics of a general peritonitis the history of the attack may throw light upon whether the peritonitis is the result of a ruptured appendix, a perforated gastric or duodenal ulcer, a cholecystitis, a pancreatitis, or intestinal obstruction. It is always an extremely difficult task to make a diagnosis of phlegmonous and gangrenous cholecystitis, as distinguished from peritonitis due to other causes, such as those already named.

(5) *Chronic cholecystitis,* in its clinical manifestations, closely resembles cholelithiasis. The chief diagnostic points are attacks of pain as in gall-stones, the presence sometimes of a pear-shaped enlargement of the gall-bladder, the absence of pain or tenderness during the intervals of attacks, and the fact that jaundice is much less likely to occur than in attacks of gall-stone colic; if it does, it is usually slight in degree. In chronic ulcerative cholecystitis gall-stones are usually present.

Complications.—(1) *In acute catarrhal infective cholecystitis* there may be vomiting, and the peritonitis that sometimes occurs may cause paralysis of the hepatic flexure of the colon and distention with gas. The lower portion of the right lobe of the liver may become somewhat elongated and cover the gall-bladder, so that the latter and not the former is palpable. Infecting organisms may be absorbed from the gall-bladder and produce endocarditis or inflammation of the urinary tract.

(2) *The complications of membranous cholecystitis* are practically the same as those present in most cases of cholelithiasis, and include intestinal volvulus and perforation of the gall-bladder.

(3) *In suppurative cholecystitis* the chief complication is perforation. This may occur into the peritoneal cavity and set up a general peritonitis. In other cases adhesions may have formed between the gall-bladder and other viscera, and the perforation leads into the small intestine, colon, or stomach. In some cases a sub-diaphragmatic abscess may result, or a communication may be established through the diaphragm with a bronchial tube. Instances have been recorded where the enlarged and suppurating gall-bladder emptied into the vagina, the urinary bladder, or the pelvis of the right kidney. The perforation may take place into the substance of the liver. The gall-bladder suppuration may be accompanied by suppurative cholangitis, which may produce abscesses in the liver and spread infection to the lungs and other parts of the body. The appendix may become infected.

(4) *The complications to be feared in phlegmonous and gangrenous cholecystitis* are jaundice from obstruction in the biliary passages, the destruction of the gall-bladder wall, the setting up of peritonitis, and the causation of a general toxemia.

(5) *Chronic cholecystitis* through the absorption of infection may cause arthritis, phlebitis, or myocarditis.

Sequelæ.—One form of cholecystitis leads on frequently to another form. Thus the catarrhal may become suppurative, and this, in turn, phlegmonous or gangrenous. The milder forms of cholecystitis may establish an empyema of the gall-bladder, especially if the biliary ducts become occluded. If the cystic duct is closed by a gall-stone or inflammatory thickening, the gall-bladder may become distended with a thin serous fluid. Ulceration may occur in any form of cholecystitis, and the simple acute form may become chronic, ending in sclerosis and contraction of the gall-bladder walls, chronic ulceration of its mucous membrane, the formation of gall-stones, chronic empyema, and the formation of many adhesions. The gall-bladder may become obliterated and its walls calcified or infiltrated with cholesterin. In chronic cholecystitis a large gall-stone may completely fill the gall-bladder.

Clinical Varieties.—Though the several forms merge into one another, for convenience of study the classification has been made of dividing inflammation of the gall-bladder into the acute catarrhal infective, the acute membranous, croupous, or fibrinous, the acute suppurative, the acute phlegmonous and its immediate sequel, the gangrenous, and the chronic forms such as the chronic catarrhal, the sclerosing, the chronic empyematous, chronic ulcerative, actinomycotic, syphilitic and tuberculous.

Treatment.—(1) *Acute catarrhal infective cholecystitis* demands **rest in bed.** The patient should be placed on a **light, nourishing and easily digested diet.** The local application of **heat** may relieve the pain, or **dry cupping** or a few **leeches** may be employed. **Morphin** should be used very sparingly and only small doses ordered. Nausea and vomiting are best relieved by **bismuth** in some form—grains 15 (1 gram) of the carbonate, by the use of **effervescing drinks,** by **dilute hydrocyanic**

acid, minims 3 (0.2 c.c.), by **cocain,** grain 1/10 (0.0064 gram), by **morphin hypodermically,** grain 1/20 (0.0032 gram). The bowels should be regulated by some mild aperient, as a **rhubarb pill,** a **saline,** or small doses of **calomel, Carlsbad water or salts, or hunyadi water.** The stomach and duodenum may be improved by a **soda and rhubarb mixture or other stomach tonics. Urotropin,** grains 5 (0.325 gram), or **salicylate of soda,** grains 5-15 (0.325-0.972 gram), may be ordered with the object of increasing the flow of bile and disinfecting the biliary channels to prevent upward extension of the infection. **Ipecacuanha** is a reliable liver stimulant, for the purpose of increasing the flow of bile. It may be given in doses of grain ¼-1 (0.016-0.065 gram). **Succinate of soda,** grains 5-15 (0.325-1 gram), every three hours, and **methylene blue,** grain ½-1½ (0.032-0.097 gram), have been lauded by some for the same purpose. **Diaphoretics and diuretics** are required to keep the skin and kidneys active. As, if jaundice is present, the bile must be eliminated by the kidneys, attention must be given to them. **Warm baths** are valuable aids, and large **warm water enemata** daily will keep the bowels clear and are thought to stimulate contraction of the gall-bladder and remove obstruction to the flow of bile. **Colon bacillus vaccine** has been employed with good results. **Drinking copiously of water** should be encouraged. If there is jaundice the administration of **ox-gall and pancreatin,** in pill or capsule, will assist in the digestion of fats.

(2) *Membranous cholecystitis* must be treated as the symptoms require. During an attack of pain this must be relieved, and **morphin hypodermically** may be required. If the attacks of pain recur, the gall-bladder should be opened and drained. Many now favor the extirpation of the gall-bladder. If it is decided to perform a cholecystotomy and drain the gall-bladder, all adhesions that may be found should be broken down.

(3) *For suppurative cholecystitis* the medicinal and general treatment is the same as for the acute catarrhal form. The treatment is, however, **surgical** and consists in opening and draining, or preferably, removing the gall-bladder. This is also true of empyema.

(4) *Phlegmonous and gangrenous cholecystitis* demand **active surgical measures.** Though the pain may be relieved by **morphin and hot applications,** no time should be lost in this way, and morphin should not be given until the diagnosis has been made. As the tissues of the gall-bladder are very seriously damaged, and often there is perforation, the operation of choice is its complete removal—a **cholecystectomy.**

(5) *Chronic cholecystitis* is treated by inducing the patient to **drink water,** by living on a **light and easily digested diet,** by the **careful regulation of the bowels,** and the employment of **urotropin** and **sodium salicylate** or other drugs that stimulate the flow of bile. **Aspirin,** grains 5-10 (0.3-0.6 gram), repeated, may relieve pain; or minims 20 (1.25 c.c.) **spirits of ether** in 4 drams (15 c.c.) of **chloroform water** every fifteen minutes is also useful. A large drink of **hot water** has given

ease. Sometimes a **hypodermic injection of morphin** is necessary. Indigestion must be treated to prevent further irritation of the biliary structures. Opening the gall-bladder and removing the mucous membrane has been practiced, but some surgeons do not regard this operation with as much favor as that of cholecystectomy.

Prognosis.—(1) *In the mild forms of acute catarrhal cholecystitis* the prognosis is good, and the attack is usually of short duration. But these attacks are prone to repeat and lead to the formation of gall-stones. The pain in recurrent attacks may be quite severe. The possibility of ulceration and perforation must be borne in mind and adds an unfavorable note to the prognosis.

(2) *The prognosis in membranous cholecystitis* depends, to a great extent, upon the completeness of the obstruction in the cystic or common duct caused by the fibrinous casts, or the presence of a gall-stone. If the obstruction induces jaundice the case may demand operative measures.

(3) *Acute suppurative cholecystitis* has little tendency to recover spontaneously. The pus does not readily escape from the gall-bladder, as the cystic duct is liable to be occluded by a calculus, by inflammatory thickening, by adhesions, by new growths, or by stricture. If the obstruction is not complete the pus may escape. When the attack is not very acute, and is obstructive enough to prevent fluid passing along the duct, the condition of empyema results. In acute cases the walls of the gall-bladder are soon invaded and a local or general peritonitis results. In such cases perforation may occur. The prognosis is bad if there is extensive peritonitis, but is more favorable when the suppuration is limited in extent.

(4) *The prognosis in phlegmonous and gangrenous cholecystitis* is very grave. The inflammation is so rapid that adhesions do not form, and the infective inflammation spreads rapidly, soon involving the general peritoneal cavity.

(5) *Chronic cholecystitis* varies in prognosis with the causation, tuberculous and malignant cases being very unfavorable. In the chronic catarrhal, sclerotic, or empyematous forms the removal of the gall-bladder or its efficient drainage holds out good prospects of a cure.

Pathology.—(1) *In infective catarrhal* inflammation the coats of the gall-bladder are swollen and thickened, and often much infiltrated. The serous coat is dull in appearance and is covered with lymph. The cystic duct is frequently occluded by inflammatory swelling, or by stricture from ulceration, or by a gall-stone. The mucous membrane may be ulcerated, and is covered with mucus or bile. The distended gall-bladder may contain serofibrinous bile-stained fluid, or, if the cystic duct has been impervious for a considerable time, the contents may be clear, like serum. It may also contain gall-stones and inspissated bile. The lymphatic glands along the cystic and common ducts are often much enlarged and hardened. The mucous membrane is villous, its glands are inflamed, the epithelium is desquamated, and the vessels are engorged.

(2) *In membranous cholecystitis* there is formed a network of fibrin which contains bile-pigment, epithelial débris, crystals and cells. When this is removed there is no trace of mucous membrane left. It is structurally quite different from the mucous membrane.

(3) *The pathology of suppurative cholecystitis* reveals an enlarged gall-bladder; the serous covering is coated with lymph, and lusterless or reddish; frequently adhesions are found; the inflammation shows a tendency to spread to other organs; coils of the intestines may be in a state of paralytic distention; the walls of the gall-bladder are swollen, softened and easily torn. The mucous membrane may be destroyed in many places, or present an uneven, reddish aspect covered with mucus and lymph. There is extensive cell infiltration into its walls. The pus is often blood-stained and gall-stones are sometimes found as part of the contents. In cases that pass on to the condition of chronic empyema, the walls of the gall-bladder become markedly thickened from inflammatory infiltration and organization.

(4) *The morbid changes found in phlegmonous and gangrenous inflammation* are similar to those in suppurative cholecystitis, but more intense. The gall-bladder is dark-colored, sometimes greenish, and greatly swollen. There is a process of rapid necrosis and sloughing. The gall-bladder is filled with a quantity of pus, blood, mucous membrane shreds and bile. Gall-stones are often present. The cystic duct is occluded either by a calculus or by thick débris from the gall-bladder, or by both. In most of these cases calculi are found.

(5) *In chronic cholecystitis* the gall-bladder wall is thickened by a fibrosis in the connective tissue, and this new formation of tissue may be considerable in amount. There is usually much cell infiltration. There is also some edema, and the mucous membrane may be replaced by a good deal of scar tissue. The gall-bladder is sometimes greatly contracted and its cavity almost obliterated—cholecystitis obliterans. At other times its wall is much calcified. Calculi may be found embedded in the wall. The mucous membrane is often quite rugose. If the gall-bladder is distended its walls are usually thin. The mucus therein contained may be very thick and is sometimes in clumps.

Historical Summary.—In 1829, Louis and Andral noted the fact that cholecystitis occurred in some of their cases of typhoid fever. Murchison, in 1884, emphasized this fact. Richard Powell, in 1818, described cases of colic, followed by jaundice, and mentioned the membrane found in the feces.

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CARCINOMA OF THE GALL-BLADDER

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Etiology.—An outstanding fact in the etiology of cancer of the gall-bladder is the frequency with which gall-stones are found in connection with it. In a large series of cases gall-stones were present in 84 per cent. In some cases in which gall-stones were not found they may have been passed. A careful examination of the facts shows that the gall-stones are the cause of the cancer and not a result. In cases of obstruction of the ducts with calculi and the development of jaundice, the percentage of gall-bladder cancer is comparatively low. It has been estimated that about 14 per cent. of gall-stone cases become cancerous. This would show that there must be some other factor than the calculi. As an explanation it has been suggested that in cases where the calculi are rough and irritate the gall-bladder the percentage of malignant growths is large.

Women are affected in the ratio of 3.7 to 1 male. This has been thought to be due to the wearing of corsets, causing pressure upon the gall-bladder and enabling the calculi to cause greater irritation. But it should be remembered that women are the victims of gall-stones about three times as frequently as men, and this of course would explain the matter.

Collected statistics show that men and women suffer at the same age. The average age of those taken ill is about 58. The disease is rare under 40. This corresponds with the calculous age. Few persons have gall-stones under 40, and after many years of cholelithiasis cancer makes its appearance.

Symptomatology.—Clinical History.—The symptoms are those arising from the cause—partly from the diseased condition of the gall-bladder, and partly from the invasion of other structures. The early history of cancer of the gall-bladder is mainly that of gall-stones, but sometimes the gall-stones do not make their presence manifest. Early in the disease there is a feeling of discomfort or fullness or pain in the right hypochondrium. There may be cutaneous tenderness at the eighth dorsal segment. There may be attacks of pain similar to biliary colic, and usually there are gastro-intestinal disturbances. As the growth extends into other organs the symptoms become modified accordingly. In time the liver and bile-ducts become involved, and jaundice is present in most instances. Ascites is met with in one-fourth of the cases. Edema of the legs may arise from interference with the venous circulation. There are finally manifestations of wasting, and the condition of cholemia develops.

Physical Findings.—In more than half the cases a tumor can be made out. At first it is smooth and of the shape of the gall-bladder; later it tends to become nodular, irregular, hard and tender. Unless bound by adhesions, it moves with the liver in respiration. If the liver becomes involved it will be enlarged and uneven. The growth may extend into the portal fissure and lead to obstruction of the portal vein; or there may be extension to the head of the pancreas. Palpation and percussion may reveal the presence of fluid in the abdominal cavity. If the bile-ducts become obstructed a progressive and increasing jaundice results.

LABORATORY FINDINGS.—If jaundice is present bile coloring matter and bile acid will be found in the urine. In cases of doubt in diagnosis the Wassermann and tuberculin tests will help to clear up the situation.

Diagnosis.—When a hard and irregularly shaped tumor, steadily increasing in size, is found in the gall-bladder in a person who has reached the age of forty years or over, the probabilities are that the new formation is cancerous. If secondary deposits can be found, the diagnosis becomes certain. When such a tumor is found in the gall-bladder, search should be made for a primary origin elsewhere in the body. A skiagram may be taken, or the patient screened.

DIFFERENTIAL DIAGNOSIS.—In the early stage of cancer of the gall-bladder the resemblance to *gall-stones* is close. This is all the more so, as most cases of cancer have a prior history of gall-stones. If the enlargement is progressive and nodular, the likelihood is that it is malignant. On the other hand, chronic cholecystitis may render the gall-bladder very indurated; and firm adhesions to the colon, omentum or stomach, with enlarged glands, may add to the doubt. Gall-stones in the bile-ducts may lead to doubt, as they may closely resemble the nodules of malignant disease. An exploratory opening is the only means of removing all doubt in some cases. *Malignant disease of the stomach, colon, head of the pancreas, bile-ducts, or the liver,* may closely simulate primary cancer of the gall-bladder: *syphilis and tuberculosis of the liver* may sometimes give rise to doubt. *A floating kidney* causing jaundice has led to a mistake in diagnosis.

Complications.—The cancerous gall-bladder may become infected with pus-producing organisms, and give rise to hepatic infection and abscess. Adhesions may form and a localized abscess may form; or the gall-bladder may perforate into the colon, or the general peritoneal cavity, causing peritonitis. General septic infection has resulted from the infected gall-bladder, and endocarditis has been reported. The disease may extend to the pylorus and colon and cause obstruction. When a communication is established between the colon and gall-bladder by adhesions and a perforation, infective cholangitis may result, and the infection may extend into the pancreatic duct, giving rise to a pancreatitis.

Association with Other Diseases.—Cancer of the gall-bladder is very frequently associated with gall-stones, and the latter are regarded as the cause of primary cancer in from 70 to 95 per cent. of all cases. In non-primary cases there is cancer in some other organ, as the rectum, uterus, stomach, esophagus, etc.

Treatment.—*Medical Treatment.*—The medical treatment is purely symptomatic. Pain must be controlled by some preparation of **opium.** The nausea and vomiting may be relieved by the administration of **bismuth, hydrocyanic acid, oxalate of cerium, carbolic acid, creosote, morphin and effervescing drinks.** Intestinal fermentation requires careful **regulation of diet,** both in quantity and kind, the heavier and bulky foods being avoided. **Salol, beta-naphthol, menthol, thymol, sodium sulphocarbolate** and artificial aids to digestion, as **pepsin, pancreatin, ox-gall, papain,** may be tried separately or in various combinations. Dr. Bulkley's treatment should be tried. (*See* Malignant Tumors of Liver.)

Surgical Treatment.—The only operation to be considered is that for the **removal of the gall-bladder.** The mortality resulting from the operation is high, usually due to the hemorrhage. This might be lessened by the administration of a few large doses of **calcium lactate,** grains 15 (1 gram), every few hours. Of those who recover from the operation, the large majority have recurrence within six months. The removal of the gall-bladder can only be expected to yield the best results when the procedure is undertaken very early in the disease. For this reason an **early exploratory opening** should be made in all doubtful cases. Operation is useless when the disease is secondary to cancer in some other part of the body, or if other organs have become involved secondarily to disease of the gall-bladder.

Prognosis.—The prognosis is very gloomy. By timely surgical treatment the life of the patient may be prolonged, and some of the symptoms ameliorated. The prospect of cure, however, is very remote.

Pathology.—The cells forming the cancer may be of the columnar, spherical or cubical type. These cells may undergo colloid degeneration, and mucoid material may fill the tubes in the mucous membrane. When the growth extends into the cavity of the gall-bladder it is usually papillomatous in form. When it invades the other layers the tendency is for the growth to be columnar and to contain a good deal of connective tissue. The cells may undergo fatty degeneration, and parts of the tumor may be necrotic. The new growth originates from the mucous membrane as a whole, including the surface epithelium and the epithelium lining the glands. The tumor may take on one of two forms, namely, growing freely from the mucous membrane as a fungating mass, or infiltrating into the walls of the gall-bladder. While the growth may commence in any part of the gall-bladder, it generally has its starting point at the fundus.

Historical Summary.—Our knowledge of cancer of the gall-bladder began when Frerichs published his work on diseases of the liver in 1861. Since then a vast amount of work has been done on the etiology, pathology, and incidence of the disease.

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ACUTE CATARRHAL CHOLANGITIS

Etiology, p. 158—Symptomatology, p. 158—Diagnosis, p. 159—Treatment, p. 159—Management of convalescence, p. 160—Prognosis, p. 160—Pathology, p. 160.

Etiology.—PREDISPOSING CAUSES.—Duodenal catarrh may readily extend into the lower end of the common duct, the ampulla of Vater and the biliary papilla. Thick mucus may also form and fill the lumen of the duct or papilla. As bile is under very low pressure in the tubes it does not require much obstruction to impede its onward flow. Conditions that cause catarrh of the duodenum are the chief factors in causing cholangitis.

EXCITING CAUSE.—Various organisms have been found, as the *Bacillus typhosus,* colon bacillus, and *Bacillus paratyphosus.* Infection of the bile-ducts may assume an epidemic form, as in Weil's disease. It may be assumed that most cases of cholangitis are infective in origin rather than a mere local catarrh.

Symptomatology.—CLINICAL HISTORY.—*Mode of Onset.*—There is usually some derangement of the digestive organs, such as loss of appetite, nausea, vomiting, coated tongue, foul breath, headache, dizziness, feeling of malaise, and, at times, pains. There may be diarrhea.

Symptoms During Progress of Disease.—*Objective Symptoms.*—The feces become clay-colored early, even before there is jaundice, and they are very offensive. Jaundice appears first in the conjunctiva, but soon spreads to the body, the facies being the earliest part to show the change, then the trunk, and lastly the legs. The jaundice does not assume the dark green color found in malignant disease obstructing the bile-ducts. The mucous membrane under the tongue and inside the lips reveals the yellow color if the blood is pressed out of it. Vomiting may continue and become quite severe. The pulse is slow—60 or less per minute—and soft. The urine is dark from contained bile, and, if shaken, yields a yellow froth. The saliva and the perspiration from the axilla sometimes are colored. At the onset there may be some rise of temperature, but later on it is normal or subnormal.

Subjective Symptoms.—The patient may complain of muscular pains. Itching of the skin, pruritus, is frequently very troublesome. Yellow vision, xanthopsia, is present. Mental lethargy or depression is not uncommon, or there may be irritability of temper.

PHYSICAL FINDINGS.—As a rule, there are no marked physical abnormalities. The gall-bladder and liver are very seldom enlarged or tender. The heart's action is slow and reduced in strength, and the pulse is of low tension and often dicrotic. When the liver is enlarged the inflammation has extended to the ducts within the liver.

LABORATORY FINDINGS.—The feces contain undigested fat and are disposed to be frothy. They contain stercobilin. The urine contains bile coloring and bile acids. There may be an excess of nitrogen in the urine, corresponding with the period in which the patient is feeling most poorly and is losing weight. Casts may be found in the urine, but rarely is albumin present. If the pancreas is involved there may be a glycosuria. In inflammatory conditions of an organ, the secretions from it may contain bile. The blood contains bile, and its serum will clump typhoid fever bacilli. The corpuscles are not altered and seldom is there any increase in the white count, though it may go up to 10,000. For the tests for bile and other abnormal constituents in the urine, *see* the article on Jaundice.

Diagnosis.—The digestive derangements preceding the jaundice, the freedom from pain, and the gradual recovery in a few weeks, are indicative of catarrhal cholangitis. It must be remembered that this disorder may occur along with other and serious liver disease. Ordinary catarrhal jaundice is met with generally among the young, and when jaundice comes on in persons after middle life there is the possibility of malignant disease. If the jaundice lasts more than six weeks catarrhal cholangitis is doubtful. If the pigmentation of the skin is very dark it is due to some other cause, such as calculi or malignant disease. If calculi is the cause of the jaundice the attacks are recurrent, marked by severe pain, fever, and increase in the icterus. When the jaundice is the result of toxemia there are evidences of some general infection.

Treatment.—GENERAL MANAGEMENT.—The patient should be **confined to bed,** with sufficient covering to ensure warmth of the surface of the body. He should be enjoined to **drink water freely; hot water** with a little **bicarbonate of soda** is an excellent beverage. **Effervescent waters** may be allowed.

The bowels must be kept open, and small doses of **calomel followed by some saline** meet the conditions well; but harsh purgation is injurious. The condition of the intestinal canal may be improved by such drugs as **salol, resorcin, beta-naphthol, bismuth salicylate,** or very small doses of **calomel.** An effort should be made to increase the flow of bile, and for this purpose **sodium salicylate, urotropin, ipecacuanha, and copious draughts of water,** will do most good. **Hot applications** over the upper part of the abdomen in the early stage of the trouble, and **massage** in the later stage are useful remedies.

Diet.—At first very little food should be given, and, until a cure is obtained, the diet should be light and easily digested, such as gruel, toast, some egg, clear soup, rice, white meat, fish, and artificial foods. Fat should be avoided. Milk with 10 grains (0.65 gram) of citrate to the pint is usually well borne.

Management of Convalescence.—During convalescence simple vegetable bitters, as **gentian, calumba, nux vomica,** combined with **dilute nitrohydrochloric acid,** will improve digestion. An excellent mixture is one containing **compound tincture of gentian, tincture of rhubarb,** and **bicarbonate of soda.**

Prognosis.—The prognosis in the large majority of the cases is good, as the recovery takes place in a few weeks without any ill results. In a few cases what appears to be ordinary, simple jaundice from cholangitis is the early stage of malignant disease of the liver or of acute yellow atrophy. At other times the obstruction in the common duct continues until serious damage is done to the liver, and what commenced as catarrhal cholangitis ends in icterus gravis with stupor, coma, and death. The longer the jaundice lasts the less favorable the prognosis becomes.

Pathology.—The changes that have been found are the following: the mucous membrane of the lower end of the common duct is swollen; there is an increase of its lymphoid tissue; and the duct may be blocked by thick mucus. The mucous membrane of the duodenum and the ampulla of Vater are swollen, as is also the head of the pancreas in many of the cases. The liver may become enlarged if the jaundice persists long enough to distend the biliary tubes and induce congestion through irritation.

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CHRONIC CATARRHAL CHOLANGITIS

Etiology, p. 161—Symptomatology, p. 161—Diagnosis, p. 161—Treatment, p. 161.

Etiology.—There are two forms, namely, those in which the inflammation is in the tubes outside the liver, and those in which the intrahepatic ducts are the seat of the disease. Chronic catarrhal cholangitis may be associated with gall-stones, cancer, stricture, pressure, parasites, and infections.

Symptomatology.—When the chronic form follows the acute, the jaundice continues, and this may vary in intensity from time to time. After attacks of fever and pain it is more marked. There may be loss of body weight. Sometimes the inflammation may spread into the tubules in the liver and cause acute yellow atrophy.

Diagnosis.—Chronic catarrhal inflammation of the bile-ducts gives rise to jaundice, which may be distinguished from hypertrophic biliary cirrhosis, as in the latter the jaundice is accompanied by an enlarged liver and spleen. In cases of cholangitis caused by calculi the pain is usually more severe than when the cholangitis is not accompanied by them, and there is a greater tendency to intermittent fever in the former.

Treatment.—MEDICAL TREATMENT.—The diet must be simple and non-stimulating. **Alcohol should be avoided. Salines and calomel** are useful in preventing constipation. **Salicylates and urotropin** may act beneficially on the biliary passages. **Drugs that aid in regulating the function of the digestive canal,** as mentioned in the treatment of jaundice and acute catarrhal cholangitis, should be tried. **A stay at some watering place** may be helpful.

SURGICAL TREATMENT.—In cases that run a prolonged course and resist the measures just mentioned, the gall-bladder should be opened and drained. This is still further justified by the fact that gall-stones may be present. By this treatment cases have been cured that were thought to be instances of hypertrophic biliary cirrhosis.

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SUPPURATIVE CHOLANGITIS

Etiology, p. 162—Symptomatology, p. 162—Diagnosis, p. 162—Complications, p. 163—Association with other diseases, p. 163—Treatment, p. 163—Prognosis, p. 163—Pathology, p. 164.

Etiology.—PREDISPOSING CAUSES.—These are local and general. Among the former may be mentioned gall-stones—the most common—parasites in the ducts, the rupture of a hydatid cyst into the gall-bladder or ducts; stagnation of the bile from any cause, such as pressure by a tumor in the neighborhood of the ducts, and the existence of a stricture. Of the general causes for suppurative cholangitis we have pneumonia and influenza through the general circulation, and cholera and typhoid fever through the portal circulation.

EXCITING CAUSE.—The exciting cause of suppurative cholangitis is some microörganism. The colon bacillus has been obtained in pure form in cultures. The *Bacillus typhosus* and *Bacillus paratyphosus* have also been found. The pneumococcus, staphylococcus and streptococcus have been isolated as the cause. The bacillus of cholera, typhoid fever, and Bacillus coli communis may pass from the duodenum into the large bile-ducts, as these organisms are motile.

Symptomatology.—CLINICAL HISTORY.—*Mode of Onset.*—This may be abrupt, but it is often slow and gradual, with malaise, rigors, nausea, or vomiting.

Symptoms During Progress of Disease.—(1) *Objective Symptoms.*—There are rigors, fever, nausea, vomiting, and depression. There will be jaundice if the biliary passages are obstructed by the inflammation, gall-stones, or other causes; and in such cases the urine will contain bile. The temperature is usually raised, but may be subnormal. There may be diarrhea.

(2) *Subjective Symptoms.*—The feeling of loss of appetite and malaise occupy a prominent place in the patient's mind. There may be considerable pain, in part due to a peritonitis of the adjacent parts. The pain may occur in paroxysms.

PHYSICAL FINDINGS.—The liver is enlarged and there may be tender conditions that progress. It feels smooth to the touch. The spleen is also enlarged. There is rigidity of the muscles over the inflamed region. Tympanites may develop.

LABORATORY FINDINGS.—The blood contains bile in such cases as become jaundiced. There is a leukocytosis, and the blood may contain microörganisms. The urine is likely to contain albumin, and, if jaundice is present, it will contain bile.

Diagnosis.—The diagnosis rests mainly on the fever and local symptoms, together with the severe constitutional disturbances present, and a steadily enlarging liver. When jaundice is present it supports the diagnosis of suppurative cholangitis as against pylephlebitis or liver abscess. In suppurative cholangitis the fever is continuous, but in gall-stone cases the fever is intermittent. In pylephlebitis the spleen is more enlarged than in suppurative cholangitis, but there is less likelihood of jaundice, and blood cultures are negative in the former and positive in the latter. In tropical abscess there would be the history of dysentery, and there might be local fluctuation. A very rapidly growing carcinoma with pressure on the ducts may simulate very closely suppurative cholangitis. Acute cirrhosis with fever, jaundice, and tenderness may be difficult to differentiate; but the history of alcoholism may throw some light upon the diagnosis. There should be no great difficulty in distinguishing malaria from this form of cholangitis, by the presence of the plasmodium in the blood.

Complications.—Local abscess may form beneath the diaphragm or in the lesser sac; or there may be leakage of pus and general peritonitis. The blood stream may become infected; microörganisms may enter the liver and be carried by the hepatic veins to the lungs or pleuræ, setting up infective inflammation in them. The infection may extend directly through the diaphragm into the pleural cavity. Arthritis, meningitis, and endocarditis may result from general infection of the blood.

Association with Other Diseases.—Suppurative cholangitis is frequently found in association with gall-stones. This relationship has been found as high as 90 per cent. The rupture of a hydatid cyst is recognized as another condition that occurs in conjunction with cholangitis. A hepatic abscess may exist first and the infective inflammation of the bile-ducts may follow. Tumors in the duodenum, by obstructing the flow of the bile and weakening the biliary ducts, favor the onset of infection of these ducts. General blood infections may precede the inflammation of the ducts, and be associated with it afterwards. Cholecystitis usually accompanies suppurative cholangitis.

Treatment.—The treatment is necessarily **surgical.** There must be an evacuation of the pus by free drainage. The tubes, the gall-bladder, and abscesses in the liver may have to be opened.

The *diet* must be easily digested and nourishing. *Pain* may call for some anodyne. **Sodium salicylate,** grains 15 (1 gram), and **urotropin (hexamethylenamin),** grains 5 (0.325 gram), are commended as the means of increasing the flow of bile and disinfecting the ducts. An **autogenous vaccine** may be prepared and used.

Prognosis.—The prognosis depends largely upon the extent of the infective process. If the case is operated upon early, while the disease is limited to the large ducts, the prospect for a recovery is very much better than if the disease has invaded the liver, the pancreas, or established a considerable amount of peritonitis. If a number of abscesses have formed in the liver the prognosis must be very unfavorable; but if there is only one liver abscess the outlook is much brighter. If the suppuration has perforated into the lung, colon or other organ, an operation and drainage will somewhat relieve the symptoms, will prolong life, and, perhaps, save life.

Pathology.—MACROSCOPICAL FINDINGS.—The mucous membrane of the affected ducts becomes swollen and ulcerated. The walls of the ducts are inflamed and thickened, and there may be peritonitis. The glands in the portal fissure are enlarged. The infection may extend into the liver along the biliary ducts, or by way of the portal veins if there is a pylephlebitis. There may be an empyema of the gall-bladder. The liver is enlarged. Around the portal spaces there are usually areas of a yellowish-green color, in which pus may form. The bile-ducts may become greatly dilated. There may be many small abscesses in the liver, or only one, or the small abscesses may become confluent into one. The pus is bile-stained. The lymph-vessels and glands may suppurate. The branches of the portal veins are likely to become infected. The pancreas may be infected and suppurate and the peritoneum covering it may become inflamed. The bile-ducts may open into the peritoneal cavity. Abscesses in the liver have extended through the diaphragm into a bronchus.

MICROSCOPICAL FINDINGS.—Microscopically there is a good deal of cell infiltration of the liver and evidence of hepatic softening. The portal veins and the large bile-ducts may be so involved in the inflammatory process as to be indistinguishable.

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The bibliography is the same as for Cholecystitis (Suppurative and Non-suppurative).

CANCER OF THE BILE-DUCTS AND AMPULLA OF VATER

Etiology, p. 164—Symptomatology, p. 164—Diagnosis, p. 165—Complications, p. 167—Treatment, p. 167—Prophylaxis, p. 167—Prognosis, p. 167—Pathology, p. 167.

Etiology.—The real causation of cancer is still shrouded in obscurity, and, consequently, in cancer of the biliary ducts only the indirect causes can be given. It has been claimed that the disease is due to the histological changes in ordinary papilloma which become malignant. While it must be admitted that sometimes benign growths may become malignant, it must be a very rare event for such a change to take place in a papilloma of the bile-ducts. Gall-stones are present in 34 per cent. of primary bile-duct cancer, but whether as a cause, or merely as a coincidence, cannot be affirmed. Males and females suffer in the ratio of 69 to 31 per cent. As to age it is practically the same as in cancer of the gall-bladder, namely, an average of 56 years. It is very rare under 50, but has been met with as early as 30, and may occur as late as 90 years of age. The passing of calculi is probably through its irritation of the duct. The calculi may have escaped into the intestine.

Symptomatology.—CLINICAL HISTORY.—*Mode of Onset.*—The onset is gradual; but there may be a sudden increase of the symptoms caused by inflammatory action or closure of the bile-ducts.

Symptoms During Progress of Disease.—(1) *Objective Symptoms.*—Jaundice is present in all cases where the cancer involves the common or hepatic ducts. The tongue is coated, there is vomiting, constipation or diarrhea, and the feces are pale, though they may contain some bile; and there may be blood in them. The pulse is not slowed as in ordinary jaundice, and the temperature is seldom raised and is more usually subnormal. The jaundice may become very deep, almost black. The urine contains bile and is dark in color. Toward the end there may be coma and delirium. There is progressive loss of weight.

(2) *Subjective Symptoms.*—The patient has a feeling of malaise and a distaste for food, especially fatty articles of diet. There may be attacks of biliary colic, due to the presence of calculi, to a blocking of the bile-passages by thick secretions and bile, or to the pressure of the new growth. The patient complains of pain, usually toward the right of the midline, but it may be felt on both sides. Itching is frequently present, and may be very intense. It may precede the jaundice, and is sometimes absent, though the jaundice is severe. Xanthopsia is occasionally present.

PHYSICAL FINDINGS.—*Observation.*—The appearance of ill health, the deep jaundice, and the loss of weight direct the attention to malignant disease of the liver or bile-tubes.

Palpation and Percussion.—The pulse is not slowed. In a large number of the cases the gall-bladder is found to be enlarged. To this there is the exception when the cancer is in the hepatic duct, the cystic and common ducts having escaped. The gall-bladder feels smooth unless the cancer also involves it. The liver is usually enlarged from distention with bile; but the enlargement may be concealed by ascites or tympanites. It is sometimes of normal size. Ascites, found in about half the cases, is caused by obstruction to the portal vein, or by irritation of the peritoneum. The spleen is enlarged in about one-fifth of the cases. The new formation in the biliary tubes is seldom palpable.

LABORATORY FINDINGS.—The urine and the blood contain bile. Albumin may occasionally be found in the urine; but there may be casts due to the irritation of the bile. Blood may be discovered in the stools.

Diagnosis.—The gradual and painless commencement, the steady increase in the jaundice, the fact that the patient's age is usually over fifty, and the absence of any discoverable cause strongly suggest *cancer of the biliary ducts. Obstructive jaundice,* however, from other causes must be considered before coming to a definite opinion.

In the case of *cancer of the head of the pancreas* the pain is more distinctly epigastric than when the biliary ducts are involved. In the case of *cancer of the pancreas,* secondary invasion of the liver is more common than when the disease begins in the ducts; but in the latter case the liver tends to become more enlarged than when the pancreas is the primary seat of the disease. In the pancreatic form the tumor can sometimes be felt; in the bile-duct form, very rarely. Cancer of the pancreas is more frequent than of the bile-ducts. The Cammidge test is helpful by determining the fat in the feces. In pancreatic cancer the saponified fat is less than the unsaponified, while in bile-duct cancer the condition is reversed. In cancer of the bile-ducts the feces contain stercobilin, but in the pancreatic disease there is none.

The distinction between *calculi in the ducts* and cancer of the ducts is made mainly on the history of hepatic colic in the former, also on the following facts: Jaundice in such cases runs a slower course than cancer of the ducts; death is usually due to some complication, as suppuration, while in cancer it is usually caused by cholemia; as the case progresses the obstruction from a calculus tends to lessen, while that from cancer increases; in obstruction from a gall-stone the gall-bladder is not enlarged, whereas in cancer of the ducts it is enlarged; finally, in calculous cases there is intermittent fever, and in cancer cases no fever occurs unless there is infection, when it is of the continuous type.

In *cancer of the liver* there is enlargement of the organ; it is infiltrated with new growth; there is an absence of jaundice, or it is not so marked as when the disease is in the ducts.

Hypertrophic biliary cirrhosis is differentiated from cancer of the bile-ducts by the facts that in the former the disease is usually met with in the young; the spleen is considerably enlarged; the liver is much larger than in cancer of the ducts; and the case is much more chronic than is the latter.

Cancer of the gall-bladder may be distinguished from the same disease in the bile-ducts by the absence of jaundice, unless the ducts become involved. When the disease is in the gall-bladder a tumor can be felt, but not so when it is in the ducts. This tumor is hard and uneven, and differs from the larger, smooth gall-bladder, the result of cancer in the ducts. If the disease in the gall-bladder extends to the bile-ducts, the symptoms of obstruction become added to those caused by the disease in the gall-bladder.

Catarrhal jaundice may be distinguished from cancer of the bile-duct by the mode of onset and the subsequent course. In catarrhal jaundice the illness comes on suddenly with vomiting, diarrhea, and gastro-intestinal disturbance, whereas cancer of the duct comes on gradually and without marked symptoms. Moreover, catarrhal jaundice disappears in a few weeks, but the cancer obstruction steadily grows worse, the jaundice deepens, and the patient loses flesh and strength. When the cancer is in the ampulla of Vater the jaundice is intermittent and the feces are normal at times. The jaundice lessens or disappears in the intervals. Attacks of fever recur with cholangitis. Diarrhea is apt to occur from time to time.

It will also be necessary to distinguish the jaundice caused by cancer of the bile-ducts from jaundice caused by tumor in the hepatic fissure, by aneurysm, by hydatids, by adhesions, or gumma. There will usually be enough in the history and physical conditions to permit of a differential diagnosis.

Complications.—The malignant new formation may become infected and suppuration result, causing metastatic abscesses in other parts of the body. There may be rupture of the duct and a general or localized peritonitis. The endocardium may be infected from the blood stream. Thrombosis of the portal vein has been noted.

Treatment.—PROPHYLAXIS.—The preventive treatment should receive careful consideration. Thorough attention, both medical and surgical, should be given to gall-stones, with the object of their prevention and removal, as a very high percentage of malignant tumors of the gall-bladder and bile-ducts has been traced to their presence.

MEDICAL TREATMENT.—The medical treatment consists in dealing with the symptoms as they arise. The *diet* must be selected so as to furnish the requisite nourishment in a digestible form. *Pain* must be relieved by an opiate, and *constipation* corrected by a suitable laxative, such as **magnesium sulphate, cascara, castor oil capsules,** or a mild pill or tablet containing **aloin, belladonna, cascara, strychnia, or other aperient.** Care must be given to the intestinal canal to prevent *putrefactive processes,* by the use of **beta-naphthol, thymol, guaiacol, calomel, or sulphocarbolate of zinc.** The *pruritus* may be relieved by **calcium lactate or chlorid** in gram doses (grains 15) several times a day. Small doses of **chloral hydrate** are sometimes helpful for this condition, the dose varying from grains 5-10 (0.325-0.65 gram) every three or four hours. *Internal hemorrhage* may be somewhat controlled by the **calcium salts. The intravenous or interstitial administration of salines** is useful as a means of warding off or relieving cholemia.

The **non-meat and potash treatment** of Dr. Buckley may be given a trial. (*See* Malignant Tumors of the Liver.)

SURGICAL MEASURES.—When the obstruction is in the common duct, the gall-bladder and small intestine may be put into communication with each other—a cholecystenterostomy. This will prevent the bile being dammed back upon the liver, and will materially aid digestion. Sometimes a fistula has been established between a dilated hepatic duct and the outside abdominal wall. The object of these operations is to prevent the occurrence of a fatal cholemia. More radical operations have been performed a number of times with the object of removing the growth. Resection of the hepatic, cystic and common ducts has been successfully carried out.

X-RAY THERAPY.—Treatment by *x*-rays has been tried, but with very doubtful results. It may restrain the rapidity of growth or lessen some of the symptoms, but it cannot cure. It may yield better results in those cases where a radical operation has been performed, and the object is to obviate a recurrence of the disease.

Prognosis.—The prognosis is very unfavorable, most of the patients dying in six months or less after the commencement of the jaundice. Death is generally caused by cholemia.

Pathology.—At least in 68 per cent. of the cases the growth is in the common duct; in 26 per cent. it is in the hepatic duct or one of its branches, and in 6 per cent. the growth is in the cystic duct. These locations account for the fact that jaundice is such a constant feature, as only those could escape where the disease is limited to the cystic duct alone.

MACROSCOPICAL FINDINGS.—Macroscopically the tumors are small and feel hard. They spread along the ducts, and, regardless of the point of origin, may involve more or less completely all the larger ducts. The growth may extend into the head of the pancreas. The growths may infiltrate the tissues, especially in the portal fissure, and project from the mucous membrane of the ducts, assuming a villous form, often modified in appearance by ulceration. These growths may surround the tubes and constrict them, or they may convert them into rigid masses. In some cases the lumen of the duct is occupied by the tumor. As the disease extends along the ducts and involves several of them, or is found in the fissure, it may be impossible to determine the exact starting point. Cancer located in the bile-ducts close to the liver resembles cancer of that organ; when it exists in the cystic duct it takes on the characteristics of disease in the gall-bladder, and when at the end of the common duct it resembles disease of the pancreas. The bile-ducts may be contracted in places or greatly dilated, and filled with bile or a rather clear mucus. If the common hepatic duct is occluded, the gall-bladder will be small, but if the occlusion is in the cystic or common duct it will be distended. Primary cancer of the bile-ducts usually destroys life by the production of cholemia before secondary deposits have appeared, so that as a rule they are absent or not found to any great extent. The intrahepatic biliary ducts become dilated and filled with bile or mucus.

MICROSCOPICAL FINDINGS.—The microscopical appearances are usually definite. When the cancer develops from the bile-ducts it is of the columnar type; if it is from the pancreas the cells are spheroidal. This constitutes a valuable diagnostic feature in deciding the origin when both the ducts and the pancreas have become involved. The microscope reveals the fact that primary malignant disease of the bile-ducts is always cancerous. In cases that are growing rapidly there is a tendency for the columnar type to change to the spheroidal type of cell; but the columnar form of cell is found in at least 87 per cent. of the cases. The spheroidal cells come mainly from the mucous glands in the ducts. Squamous cells may also be seen as the result of proliferation of the surface layer of the mucous membrane. The fibrous tissue of the growth is plentiful and well organized, giving these tumors their hardness. As the result of the biliary obstruction a certain amount of cirrhosis may sometimes be found in the liver; the bile capillaries may be degenerated in places and new ones formed in other parts; the liver cells may be undergoing necrosis and regeneration at the same time; there may be necrosed areas, and, in places, the liver cells may be bile-stained, even deep green in color.

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ABNORMALITIES OF THE LIVER, GALL-BLADDER AND BILIARY DUCTS

Lobulation, p. 169—Accessory livers, p. 169—Furrows, p. 170—Deformed liver, p. 170—Riedel's lobe, p. 170—Displacements of the liver, p. 170—Hepatoptosis, p. 171—Abnormalities of the gall-bladder, p. 172—Obliteration of the biliary ducts, p. 172.

Lobulation.—The liver may not be divided into lobes; at other times it is irregularly lobulated, and the organ considerably subdivided into portions. This lobulation of the liver is probably the result of some disease process, such as tuberculosis, syphilis, or intra-uterine peritonitis.

SIZE OF LOBES.—Sometimes the left lobe is much larger than the right lobe, the latter being reduced in size inversely as the left one is enlarged. This variation is thought to be due to some change in the circulation in the different portions of the liver.

PROJECTING LOBES.—Not infrequently portions of the liver project from its surface. These finger-like processes are of no significance. They are usually found on the under aspect of the right lobe. They may be joined to the liver by such a narrow band of tissue as to be distinctly pedunculated.

Accessory Livers.—Separate portions of liver tissue have been found in the suspensory ligament. A pedunculated or accessory lobe may become detached and form an isolated portion. Long-continued pressure, as in tight lacing, may detach a portion of the liver by atrophy and pressure.

Furrows.—Well-marked furrows may be formed on the surface of the liver by pressure from the ribs or diaphragm. The furrows due to pressure from the former follow the course of the ribs on the surface of the right lobe, while those due to pressure from the diaphragm run in an anteroposterior direction, varying in number from one to six. They are generally found in persons who have suffered from bronchitis or emphysema, or who have worn tight corsets.

Deformed Liver.—The wearing of tight corsets sometimes forces the liver downward into the abdominal cavity, and a larger portion of the surface of the right lobe comes into contact with the abdominal wall, and this elongated portion covers the viscera. A part of the right lobe may become greatly lengthened, or even separated from the body of the liver except for the fibrous capsule. On the other hand, the liver may be forced upward, and become deformed by pressure against the vertebræ. Portions of the liver that have been deformed by pressure are liable to become the seat of disease, because of the impaired circulation and the atrophic changes that occur.

Riedel's Lobe.—Several names have been given to this condition, such as *linguiform lobe, partial hepatoptosis,* or *appendicular lobe.* The two main causes are cholelithiasis and badly fitting tight corsets. This accounts for the frequency of this deformity in women. These elongations of the liver are usually from the under surface of the right lobe. On palpation the tumor may be felt; it moves with the liver in respiration. The symptoms in these cases are usually such as arise from the cause, such as the dyspepsia from tight lacing, or the colic and jaundice of calculi. These abnormal lobes may give rise to symptoms by pressure on the bile-ducts or other structures. The treatment is directed toward the cause, as it is but seldom that the lobe has to be removed or fastened to the abdominal walls.

Displacements of the Liver.—There are many influences that act upon the liver so as to cause its displacement. The liver may be abnormally situated congenitally, as in transposition of the viscera or of the liver only. The abdominal walls may be deficient, permitting the protrusion of the gland, a condition known as hepatocele, or hepatomphalos. Due to a defect in the diaphragm, one or the other lobe of the liver may project into the chest. Injury to the diaphragm in adult life may be followed by protrusion of the liver into the thorax. Curvature of the spine or an irregularly shaped chest, a large accumulation of fluid or air in the right pleural cavity, tumors in the lung and mediastinum, emphysema, and pericardial effusion may displace the liver downward. A subphrenic abscess on the right side, or peritoneal adhesions, may have a similar effect. The liver may be displaced upward by ascites, abdominal tumors, or distention of the colon or intestines by gas. A pancreatic cyst or a tumor of the right suprarenal body or a retroperitoneal growth may press it forward, and distended intestines, in front of the liver, and acute yellow atrophy cause its displacement backward. Free gas in the abdominal cavity, from a perforation of the stomach or bowel, has the same effect. The treatment is directed toward the cause.

Hepatoptosis.—This condition has been spoken of as movable liver, wandering liver, and prolapse of the liver. Minor degrees occur in about 15 per cent. of those suffering from digestive disorders; but in its complete form it is not frequent. In a group of 216 cases, 180 were females and 36 were males, or 5 to 1. Most of the cases occur after forty years of age.

ETIOLOGY.—The liver is maintained in position by the peritoneal ligaments, namely, falciform, coronary, and lateral; by the inferior vena cava attaching the liver to the diaphragm; and by the abdominal muscles maintaining the viscera in their place. Any state of ill health or other conditions that weaken the suspensory ligaments and abdominal muscles will favor the production of hepatoptosis. Among the causes may be mentioned tight corsets, frequent pregnancies, general debility, anemia, ascites, tumors, fat, flatulence, and chronic constipation. There may exist a general state of visceroptosis. Injuries, violent exertion, coughing, vomiting, or straining may cause the condition to develop suddenly in those predisposed to it.

SYMPTOMATOLOGY.—*Physical Findings.*—The liver may be rotated so as to bring its upper surface in contact with the abdominal walls and carry its anterior border down into the abdominal cavity; or the liver may be rotated so as to cause the right lobe to descend, reaching even the iliac fossa. The liver may form adhesions, rendering its replacement difficult; or the displacement may be associated with cirrhosis, gall-stones, cancer, cholecystitis, and cholangitis. The liver may be very movable, especially if its ligaments have become quite relaxed. It may be possible to move it about freely in the abdominal cavity, and it may change its position with a change in the position of the patient. It may be possible to rotate it on its axis. A movable liver may cause pain, vomiting, hemorrhage, colic, colitis, jaundice, ascites, dyspepsia, cough, dyspnea and nervous disorders. At times there may be no symptoms.

DIAGNOSIS.—The diagnosis rests mainly on the shape of the tumor, on the fact that it can be replaced into the site of the liver, and that when the tumor is down in the abdomen, the liver region is flattened and resonant on percussion. Care should be taken to distinguish it from cancer, hydatid cyst, gall-stones, or tumors of the omentum. From malignant disease it can be distinguished by the condition of the patient in the latter, the existence of jaundice and the irregular shape of the liver, and the ease with which the movable liver can be replaced. In the case of hydatid cysts the tumor usually projects from the upper surface of the liver, there is an absence of free movement, and the liver dullness remains normal. The floating liver may also be accompanied by gall-stones, and some difficulty in diagnosis may arise; but the movability of the liver should enable one to make the distinction. Tumors and cysts of the omentum or mesentery may be somewhat movable, but they cannot be pushed into the site of the liver, and the liver dullness is maintained.

TREATMENT.—*General Management.*—In the first place, **improper methods of dressing** must be **corrected.** **Tight lacing** must be **forbidden** and **badly fitting corsets** must be **replaced** by those of suitable make; straight front ones are the best. Some appliance should be adjusted to the lower part of the abdomen to support the muscles and raise the viscera. For this purpose some form of **bandage or belt** is used. It should fit well and extend as low down as the hips, and embrace the lower half of the abdomen. Such an apparatus gives the liver much support. The belt should be put in position before the person rises.

Nourishing diet should be ordered, as these patients are debilitated and require toning. The diet should be regulated to suit the respective types of patients, such as the fat or spare, the diabetic or jaundiced, and the heavy eater.

The **bowels and digestion require regulation**; **flatulence and dyspepsia** must be **relieved**, and **suitable aperients** ordered for constipation, as it is important to guard against portal congestion.

Exercises to develop the tone of the abdominal muscles should be practiced. Stooping and then throwing the body as far back as possible, deep breathing exercises, and raising the head and shoulders without the aid of the arms, the person lying on the back, or doing this exercise with one or both of the extended legs, will be found of value. These movements should be performed six to twelve times two or three times a day. **Massage and electrical treatment** have been found useful.

Surgical Measures.—Surgical measures may become necessary. The operation of fastening the liver to the abdominal walls has been performed with excellent results. The objection to hepatopexy is that the condition of loss of tone in the ligaments and muscles still remains. Suturing the round ligament to the cartilage of the seventh rib has also been tried. Rubbing the serous surfaces of the liver and diaphragm to induce adhesions has also had its advocates. These operations may retain the liver in position for such a time as will enable the muscles to gain tone. If gall-stones are present they should be removed.

Abnormalities of the Gall-bladder.—Tight lacing by badly shaped corsets tends to force the duodenum downward. This causes tension on the cystic duct, resulting in an obstruction to the flow of mucus from the gall-bladder, and its dilatation, which may go on to a very considerable degree.

Obliteration of the Biliary Ducts.—*Etiology.*—This may follow the inflammation or ulceration in the bile-ducts caused by calculi, or infections such as typhoid fever, influenza, or syphilis. It may follow operations.

SYMPTOMATOLOGY.—The symptoms are those of obstructive jaundice.

Physical Findings.—The bile-ducts may be congenitally closed or non-developed. Some writers have held that there is a defect in the development of the ducts, while others regard the condition as the result of toxic agents reaching the fetus through the placenta and setting up a descending cholangitis in the fetus. Some of the poison may reach the liver directly along the umbilical veins, while some of it may enter the child's circulation by way of the ductus venosus and reach the liver through the hepatic artery. The liver is enlarged in almost all these cases and considerably cirrhosed. It is bile-stained, dark green in color, and uneven on the surface. The cirrhosis is of the mixed portal and biliary types, and takes on the characteristics of the unilobular and multilobular forms. There is an increase in the fibrous tissue of the liver and atrophy of the liver cells. Many of the intrahepatic ducts are dilated. The gall-bladder may be distended, but is usually contracted, thickened, fibrotic, and surrounded by adhesions. The spleen is enlarged in nearly all the cases, and very much so in some. Jaundice may be present at birth or come on soon afterwards.

DIAGNOSIS.—An accurate diagnosis can only be made by an exploratory opening. The jaundice, hemorrhages, the size of the liver and spleen, with the absence of any infection, are the chief points in the diagnosis.

TREATMENT.—If syphilis is suspected the treatment should be of a specific nature. In some cases the stricture can be resected, and in others the gall-bladder may be united with the intestine. The medical treatment is symptomatic.

PROGNOSIS.—The prognosis is most unfavorable, most of the cases dying within a few weeks, but occasionally life may be prolonged for ten months.

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TUBERCULOSIS OF THE LIVER

Etiology, p. 174—Symptomatology, p. 174—Diagnosis, p. 175—Complications, p. 175—Clinical varieties, p. 175—Treatment, p. 176—Prognosis, p. 176—Pathology, p. 176.

Etiology.—The cause of tuberculosis of the liver is the *Bacillus tuberculosis.* The most frequent channels of entry into the liver are the portal vessels. Next in frequency is the hepatic artery. The disease may be conveyed to the liver of the fetus through the umbilical veins from the placenta. In rare instances the bacilli may find their way into the liver substance from an infected condition of the peritoneum. The infection does not ascend the bile-ducts, as the bacilli are not motile and the flow of bile is away from the liver.

Symptomatology.—Seldom is any disease accompanied by fewer symptoms. The miliary form has no definite clinical manifestations apart from the general disease, of which the liver involvement is only a part. Sometimes there is jaundice. If there are many tubercles on the surface there may be pain from friction and a friction rub. When the tuberculous process involves the bile-ducts there is a remarkable freedom from jaundice, and there is no bile in the urine. There is sometimes pain, but this may arise from tuberculous disease in the intestines or peritoneum. Ascites may be caused by peritonitis or pressure on the portal vein by enlarged glands. When the disease assumes the form of caseous masses in the liver, there may very occasionally be jaundice from pressure on the bile-ducts. The spleen is sometimes enlarged. There is loss of strength and weight; but a diagnosis can hardly be made. A tuberculous focus may become infected with a pus-producing organism and an abscess may result. In such cases there may be pain, swelling, rise of temperature, peritonitis, rupture of the abscess, and an increased white blood-count. When the tuberculous disease complicates hepatic cirrhosis there is enlargement of the liver, moderate jaundice and some ascites. It has been noted in some cases that the sufferer from cirrhotic liver later on becomes a victim of tuberculosis.

Physical Findings.—These, as a rule, are rather negative. Sometimes a friction rub can be made out. In a few cases the liver is swollen and tender and an abscess forms. In a few cases the spleen is found to be enlarged. When there is cirrhosis of the liver there is some enlargement of it as well. The liver is also larger than normal in cases with considerable caseation, especially if the bile-ducts are involved to any extent.

Laboratory Findings.—The only laboratory test that need be noted is the discovery of the *Bacillus tuberculosis* in the sputum, urine, feces, or discharges or tissue from the liver if any operation is performed.

Diagnosis.—The diagnosis of tuberculous disease must depend to a great extent upon the existence of tuberculosis in some other organ, during the progress of which symptoms of liver trouble make their appearance. Tuberculous nodules on the liver can sometimes be felt.

Complications.—The caseous masses in the liver may become infected with pyogenic bacteria, causing abscess formation. Should rupture of the abscess occur, general or localized peritonitis will result. Jaundice and ascites may also appear if the disease gives rise to obstructive pressure on the bile-ducts or portal vein. When there is cirrhosis of the liver from any cause, tuberculous invasion of it is liable to produce a rapidly fatal illness.

Clinical Varieties.—(1) In the miliary form of the disease, the infection usually reaches the liver through the hepatic artery, but may also be carried to it by the portal vein. (2) Another variety is that form of the disease where localized caseous masses are found in the liver. The infection in this form of the disease is carried by the portal veins, and the tubercles are formed along the course of their branches. These tubercles tend to remain as isolated caseations. (3) There is another variety in which the infection is brought by the portal vein from the intestines; but the tubercles are first formed in the portal spaces. These tubercles become masses of granulation tissue which caseate and soften, breaking into the hepatic bile-ducts. Cavities are thus formed and an infected condition of the biliary passages established. (4) Tuberculous abscess in the liver, though regarded as a clinical variety, is really the result of the infection of a tubercle or a tuberculous cavity by some pyogenic organism. It is, therefore, a complication rather than a distinct variety.

Treatment.—The treatment is almost entirely of a symptomatic and expectant nature. In a few cases tuberculous masses have been removed from the liver, and tuberculous abscesses have been opened and drained with advantage, Mayo-Robson's case being in good health at the end of two years.

Prognosis.—The prognosis is most unfavorable. In the case of miliary tuberculosis of the liver, it is only part of a general miliary tuberculosis, and must necessarily be speedily fatal. If miliary tuberculosis invades an already cirrhotic liver the downward course of the patient is very rapid. In other varieties of hepatic tuberculosis there is prior tuberculous disease in the intestines in most cases, which removes any hope of a recovery. If the liver becomes infected from a tuberculous condition of the peritoneum, the chances for a prolongation of life are brighter than in the forms just mentioned; but the prospects of a recovery are very remote. If the child is born with a tuberculous liver due to placental infection the prognosis is decidedly gloomy.

Pathology.—MILIARY FORM.—The tubercles in this form are very small; they are at first grayish in color, but when older may become yellowish. They may be seen studding the surface of the liver. When the liver is cut it may present the appearance of cloudy swelling, as the tubercles are too small to be seen by the unaided eye. The liver is generally somewhat enlarged, is fatty, is venously engorged, there may be inflammation of the capsule, and small abscesses may be found in its substance. The tubercles are found inside the lobules, or are intralobular. They are formed at first of small round-cells, but later on giant-cells are present, which undergo caseation. These giant-cells result from the conglomeration of the small cells, or the continuous growth of cells with division of their nuclei.

SOLITARY TUBERCLE FORM.—When the infection reaches the liver through the portal veins the tubercles form in the portal spaces, between the lobules, being enclosed in a fibrous capsule, and are thus contrasted with the miliary form, where the tubercles are formed within the lobules. The tubercles are larger than in the miliary form, and in the early stage of the disease they are firm; they caseate later on. When cavities form these walls are covered with caseated débris and are bile-stained and greenish. These cavities may attain considerable size, varying from that of a pea to that of a small orange. These cavities are surrounded by a capsule composed of the fibrous tissue of the portal spaces. These cavities may perforate into the bile-ducts and discharge their contents through, mixed with the bile. This form of hepatic tuberculosis is pericholangitic, the bile-ducts becoming affected when perforation occurs. Nodules of lymphadenoma and cancer may resemble tubercle, but they do not caseate, and break down into cavities. The nodular areas in portal cirrhosis may take on some resemblance to the tubercle; the history of the case will remove doubt. Tuberculosis of the liver, softening and emptying into the bile-ducts, is comparable to tubercles in the lungs discharging into the bronchi.

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SYPHILIS OF THE LIVER

Etiology, p. 177—Symptomatology, p. 177—Diagnosis, p. 179—Complications, p. 179—Sequelæ, p. 180—Treatment, p. 180—Prognosis, p. 181—Pathology, p. 182.

Etiology.—There is only one cause of syphilis, namely, the spirochete. The place of entry into the body is very varied. In the matter of congenital syphilis the writer holds firmly to the view that the mother must become infected, and afterwards impart the disease to the fetus. It seems quite impossible for the father to infect the ovum through the spermatozoön.

With regard to *age*, the early congenital cases of syphilis of the liver occur at or soon after birth, the late congenital cases come between ten and twenty usually, and sphilis of the liver in the acquired cases appears in the great majority between thirty and fifty years of age.

Symptomatology.—The symptoms of syphilis of the liver vary considerably with the stage, and whether the condition is congenital or acquired. The symptoms will be discussed under the following headings:

Early Congenital Syphilis of the Liver.—If the infection has been severe the child may die *in utero,* be born with the symptoms of the disease, or appear healthy at birth, soon to become affected. Jaundice and ascites in these cases are usually absent, but the liver and spleen are enlarged. These features taken with other evidences of syphilis are the basis of a diagnosis. The greater the hepatic enlargement, the severer the infection. The liver is tender on pressure. When jaundice does occur it is due to pressure on the bile-ducts by enlarged glands, or granulation tissue in the liver, or to an infective cholangitis, either by the spirochete alone or accompanied by other organisms. The jaundice may assume the grave type. When ascites occurs it is the result of inflammation of the peritoneum or capsule of the liver, or to enlarged glands in the portal fissure obstructing the venous flow. Attention should be paid to the condition of the child and the presence of emanciation, anemia, debility, mucous membrane sores, skin eruptions, bony nodes, iritis, vomiting and other stigmata of syphilis. The lesions are those of secondary syphilis.

Late Congenital Syphilis of the Liver.—This form of congenital syphilis comes on during the second decade. The patient is poorly developed and bears many marks of syphilis, such as disease of the bones, organs of sense and amylaceous degeneration. The spleen and liver are enlarged, and the latter may be markedly so. The kidneys may undergo lardaceous degeneration and albuminuria, and uremia may be present. Gummata or syphilitic cicatrices may cause jaundice or ascites, by pressure on the bile-ducts or veins. The ascites may also result from peritonitis or be part of the general dropsy of amyloid disease of the kidneys. There may be serious disease of the arteries, ending in obliteration of many or some of them and the production of corresponding symptoms. The lesions and symptoms of late congenital syphilis are characteristically tertiary.

Multilobular Cirrhosis of Congenital Syphilis.—In some cases of congenital hepatic syphilis, the liver undergoes characteristic cirrhosis, and the clinical manifestations are much the same as those of portal cirrhosis in childern, namely, ascites, enlargement of liver and spleen, jaundice sometimes, and hemorrhages from the stomach or nose.

Acquired Hepatic Syphilis.—During the early part of the secondary stage and concurrently with the rash on the skin, jaundice may come on, and sometimes becomes very severe. The combination of the roseola and the jaundice imparts to the patient a very striking appearance. In this form of jaundice there is an absence of pruritus, there is seldom any gastro-intestinal disturbance, and bile is usually absent from the urine. For the etiology of this jaundice several views have been advanced. It may be due to a specific cholangitis or to syphilitic new formations in the bile-ducts. It has also been thought to be of hemolytic origin. It is more probably the result of a hepatitis and the changes this induces in the small bile-ducts within the liver.

In the tertiary stage of syphilis large gummata may form. These have been large enough to reveal their contour through the abdominal walls when the patient is lying on his back. These gummata may be accompanied by amylaceous degeneration in the kidneys. If a gumma is very large the ordinary symptoms of pressure may arise, and a gumma or syphilitic cicatrix in the portal fissure may cause jaundice. The syphilitic lesions of tertiary acquired syphilis may clinically resemble portal cirrhosis, perihepatitis, biliary hypertrophic cirrhosis, various types of liver tumor, amyloid disease, suppuration of the liver when a gumma is infected, gall-stones if the ducts are obstructed by the syphilitic processes.

PHYSICAL FINDINGS.—In many cases the physical signs and manifestations of syphilitic disease of the liver are very indefinite, and would fall short of enabling one to arrive at a diagnosis apart from other evidences of syphilis. Even in those cases with such conditions present as jaundice, ascites, or tumor in the liver, in very few instances could a direct diagnosis of syphilis be made. The history of the case must be carefully studied. The conditions that may exist are ascites, jaundice, enlargement of the liver and spleen, an uneven surface of the liver from the presence of gummata, and tenderness to pressure. In some cases of syphilis of the liver, there are no physical manifestations.

LABORATORY FINDINGS.—The laboratory study of such cases resolves itself into the finding of the *Treponema pallidum* and a positive Wassermann reaction. The spirochete may be discovered in discharges from pustular or vesicular eruptions; and the blood or spinal fluid should be tested for the luetic reaction.

Diagnosis.—It may be very difficult to make a diagnosis upon the symptoms and signs arising from the liver alone; but if these are taken in conjunction with other evidences of syphilis, the diagnosis can usually be made with certainty. A child newly born or only a few weeks old, presenting the conditions of enlargement of the liver and spleen, and perhaps jaundice, should arouse the suspicion of syphilis. This is strengthened if there is no other cause, such as tuberculosis, gastrointestinal trouble or rickets. If to this is added pustular or vesicular skin lesions, iritis or keratitis, mucous membrane sores, bony nodes, and a positive Wassermann, there need be no doubt.

Enlargement of the liver and spleen with ascites in a badly nourished and poorly developed person of ten to twenty years of age is almost pathognomonic. The discovery of lesions in the bones, deafness, keratitis, Hutchinson's teeth, and amyloid disease should confirm the diagnosis, which becomes a certainty if the Wassermann test is positive. The onset of jaundice or ascites in an adult, tenderness of the liver on pressure, the enlargement of the liver and spleen, and the discovery of tumefactions on the liver, should be ample to create the thought that syphilis is the cause. Careful search will find other evidences, all being confirmed by a blood test.

Complications.—Sometimes a softening gumma in the liver may become infected with a pus-producing bacterium, resulting in an hepatic abscess. Lardaceous disease of the kidneys may also exist, causing albuminuria, dropsy, or uremia. The jaundice of syphilitic hepatitis may pass into acute yellow atrophy if there is marked intercellular infiltration.

Sequelæ.—The after-effects of hepatic syphilis are not very numerous, but the principal ones are cicatricial contraction and obliteration of one or more of the large bile-ducts. The liver may become very much distorted in appearance from the formation of connective tissue in its substance, which undergoes much contraction, leaving deep sulci on its surface. Perihepatitis and peritonitis may produce permanent adhesions.

Treatment.—*Early Congenital Syphilis.*—The treatment of the early congenital or infantile form is best carried out by means of **inunction.** Grains 15 (1 gram) of mercurial ointment should be thoroughly rubbed into the skin once a day, into different portions of the skin each time. The axillæ, over the liver and spleen, the sides of the abdomen, and the inner aspects of the thighs are best suited for this method of treatment. This should be continued for three months, when a week or two of omission may be made each month.

During the second year the treatment may be given one month in every three. Small doses of **one of the iodids** should be given during the second and third years. During the fourth year the inunction should be omitted and the dose of the iodid increased. Of **potassium, sodium, or ammonium iodid,** the dose should be 1 grain (0.065 gram) during the first year; grains 2 (0.130 gram) during the second year; and grains 3 (0.195 gram) during the third and fourth years, exercising care to avoid gastric irritation by reducing the dosage.

Hydrargyrum cum creta (gray powder) is an excellent form of mercury for children, giving ½ grain (0.032 gram) for the first three months, then 1 grain (0.065 gram) thereafter, twice daily. For a **combined treatment with potassium iodid and mercury,** this formula answers well:

℞

Potassii iodidi	℥ i	(30 grams)
Hydrargyri perchloridi	grain i	(0.065 gram)
Aquæ ad	℥ iv	(118.40 c.c.)

Of this mixture, the dose should be minims 5 (0.30 c.c.) during the first year, minims 8 (0.50 c.c.) during the second year, minims 12 (0.74 c.c.) during the third, and minims 15 (0.90 c.c.) during the fourth year, thrice daily.

Special care should be given to the **teeth,** with the object of avoiding salivation. Good results may be obtained by the administration of

Donovan's solution (solution of iodids of arsenic and mercury), in doses of minims ½ in the first months of life, gradually increasing to minims 2 toward the end of the first year. **Salvarsan** has been given to babies, but it is safer to begin by giving a dose to the nursing mother first. The child will subsequently stand this form of treatment better. A **mercurial bath** made by putting 5-10 grains (0.0325-0.065 gram) **bichlorid of mercury** in enough warm water to cover the child's body may be given for ten to twenty minutes daily. **Mercury tannate** in doses ranging from grain 1/10 (0.006 gram) during the first year to grain ½ (0.0325 gram) during the fourth year, is a favorite with some. **Calomel** may also be employed in fractional doses of grain 1/10 (0.006 gram) to begin with. When the symptoms are urgent relief is obtained most rapidly by a **combined mercurial and iodid treatment.** During the time inunction or mercurial baths are used the **iodids** may be given internally. It is best to administer the iodids just before food.

Late Congenital Syphilis.—Cases of late congenital syphilis of the liver are treated as would be the tertiary lesions of the acquired disease. When the liver takes on the changes found in cirrhosis, the same restrictions as to foods and alcoholic drinks laid down in the treatment of portal cirrhosis (p. 98) must be enforced here. **Iodids and mercurials** must be prescribed, but the scar tissue formed may not yield to treatment.

Acquired Syphilis.—Involvement of the liver during the progress of acquired syphilis must be treated along recognized lines. **A combination of potassium, sodium and ammonium iodid,** in doses of grains 15 (1 gram), or **sodium iodid,** grains 15 (1 gram), and **ammonium chlorid,** grains 5 (0.325 gram), three or four times daily, will give good results. These doses may have to be considerably increased in some cases. Along with the iodid treatment the patient may be given **mercurial inunctions or baths.** If these are not employed, some mercurial should be given internally with the iodids; and for this purpose **liquor hydrargyri perchloridi,** dram ½-1 (1.88 to 3.75 c.c.), may be added to each dose. A good formula is:

℞
Hydrargyri cum creta......grains i or ii (0.065-0.13 gram)
Quininæ sulphatis ....grain i (0.065 gram)
Pulveris opii ....grain 1/6 (0.01 gram)
Ft. into capsules, to be taken 3 or 4 times daily.

The **intravenous treatment** by means of the arsenical preparations should be employed as indicated by the conditions.

Prognosis.—The prognosis *in the early form of congenital hepatic syphilis* will depend upon the severity and degree of advancement of the attack. If the child is born with marked symptoms the prognosis may be gloomy; but if the symptoms come on after birth the outlook is much brighter. *In the late form of congenital hepatic syphilis* the prognosis is never very good, as these patients are poorly developed and often have a seriously diseased condition of the arteries. In addition, there may be extensive lardaceous disease. In those cases in which the liver has become cirrhotic the prognosis is very bad. When the liver becomes diseased *during the progress of an acquired* case of syphilis the prospects of a recovery are good, unless the hepatitis has caused such destruction as to cause yellow atrophy, or has caused much scarring of the liver with deformity, or produced strictures of the bile-ducts.

Pathology.—*In the early congenital form* the chief morbid changes in the liver are the following: the capillaries are distended and filled with a variety of cells; there are numerous scattered foci of a yellowish color, and a distinct proliferation of the connective tissue. *In the late congenital form* of syphilis of the liver the lesion is tertiary in character, and comes under the description of the gumma. With this there is the formation of fibrous tissue, the development of cicatrices, and the occurrence of amyloid degeneration. In the cirrhotic liver there is a marked hyperplasia of the interlobular connective tissue.

In acquired syphilis the liver may reveal two main conditions. The first is one of acute inflammation with biliary capillaries engorged with cells. This may give rise to jaundice or yellow atrophy. The other morbid change is the gumma. These may be numerous, small, yellowish or pinkish masses. A number of these may coalesce in one of great size. Surrounding these gummata there are walls of fibrous tissue. This fibrous tissue may form cicatrices and greatly distort the liver. Along with these tertiary lesions there are not infrequently amyloid changes to be found in other organs, especially the kidneys.

Historical Summary.—Paracelsus, in 1510, wrote of jaundice occurring in a person suffering from syphilis. Sanchez noted the connection in the eighteenth century. Gubler, in 1853, studied the condition closely and laid the foundation for a sound conception of the ravages of syphilis on the liver. In France, Lancereau, in 1868, did excellent work on the same subject. To Sir S. Wilks, more than to any one else, is due our present knowledge of the condition of the liver in congenital and acquired syphilis. His papers appeared in the *Pathological Transactions* from about 1857 onward.

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CHOLELITHIASIS

(*Gall-stones*)

Etiology, p. 183—Symptomatology, p. 185—Diagnosis, p. 186—Complications and sequelæ, p. 186—Association with other diseases, p. 188—Clinical varieties, p. 188—Treatment, p. 189:—Prophylaxis, p. 189; Management of convalescence, p. 192—Prognosis, p. 192—Pathology, p. 193.

Etiology.—*Climate and Temperature.*—There can be no doubt that climate and temperature have some influence on the formation of gall-stones, as they are comparatively rare in hot climates, and become much more common in cold countries. Cold and damp have some influence in causing catarrh of the gall-bladder.

Sex.—Sex is an important predisposing factor, as the condition is found, when large numbers are compared, three times as often among women as among men. Females, oftener than males, are the victims of constipation, faulty methods of clothing, sedentary habits, lax abdomens, tumors, and tight lacing; pregnancy, also, is a frequent factor.

Age.—As to age, in a series of statistics, it is found that 75 per cent. of cases occur after forty years of age, and 25 per cent. under this age. The largest number are met with between fifty and sixty. Under twenty there is only about 1 per cent.

Race.—With regard to race, it may be said that negroes seldom suffer, and the same is true of the Egyptian. The natives of India are about half as prone to the affection as the white inhabitants. England and America are about equal, with approximately a 7 per cent. incidence.

The people of Russia, Sweden, Hungary, Austria and Germany yield the highest percentage of sufferers.

Heredity.—Heredity is held by some to play an important part, and by others to be quite negligible. The tendency to gout, obesity, indigestion, and indolent habits may constitute family characteristics, and afford some foundation for the belief in the heredity of gall-stones, as such persons are predisposed to the complaint.

Occupation.—Occupation plays an important rôle, as the sedentary, such as clerks, writers, certain kinds of business people, inmates of asylums, homes for the aged, and workhouses, yield a larger percentage of victims than those leading an active life.

Diet.—Diet is a factor in the production of gall-stones, more by indirect than direct influences. Errors in diet may cause intestinal trouble and, eventually, cholecystitis, the chief cause. Excessive eating is causative in this way. It has been stated by some that starchy foods favor the formation of gall-stones, while a protein diet has the opposite effect. It must be remembered that in countries where rice is the main article of diet the disease is rare; and in temperate and cold climates where more meat is consumed it is more common. The free use of fats is said to be a cause, but the excessive consumption of fats is liable to cause a catarrhal condition of the intestinal mucosa. Upon the whole, therefore, it is a question mainly of the excessive use of any food and abuse of alcoholic beverages rather than the kind. The restricted intake of water is certainly causative. In a word, everything that favors stagnation of the bile favors the production of gall-stones.

EXCITING CAUSE: THE ORGANISMS.—*Bacteriology.*—It is now admitted that the gall-bladder or intra-hepatic tubes must first become infected.

The organisms responsible for this condition are the colon bacillus, the *Bacillus typhosus,* and mild strains of the streptococcus and staphylococcus. An inflammation results, which in turn favors the formation of the calculi. It is now observed that severe and acute inflammatory processes do not give rise to gall-stones so readily as mild and chronic inflammations. Mere obstruction in the flow of the bile is not alone effective, for there must be a catarrhal inflammation and an over-production of cholesterin.

Distribution in the Body.—There has been much discussion over the route by which the bacteria reach the biliary tubes and the gall-bladder. By some they are thought to pass up the common and cystic ducts to the gall-bladder, or the hepatic ducts into the liver, in the instances where calculi have been found in these ducts. There are objections to this ascending theory, such as the bacteria making their way against the flow of the bile, and the escape of the pancreatic duct which communicates with the ampulla, as does the common duct. Others have suggested that the organisms are carried to the liver by the blood stream. This would explain those cases of calculi in the intrahepatic ducts. Bilirubin calcium calculi may form in the intrahepatic ducts and be carried by the bile into the gall-bladder, constituting the nuclei around which the cholesterin gathers. These infective inflammations lead to the excess formation of bilirubin calcium and cholesterin; and these elements, with the coloring matter of the bile, mucus and broken down cells, combine in various proportions to produce the different types and sizes of biliary calculi.

Symptomatology.—CLINICAL HISTORY.—*Mode of Onset.*—The causes leading up to the formation of gall-stones are slow processes; but the actual onset of an attack of hepatic colic is sudden and induced by a calculus passing along the cystic or common duct. The cause of the pain is either spasm of the duct or inflammation in the gall-bladder, which has the effect of starting the calculi in motion.

Symptoms During Progress of Disease.—(1) *Objective Symptoms.*—Vomiting is a very common condition, and sometimes relieves the pain. Chills will be observed in at least 66 per cent. of the cases. Jaundice will be present in 15-20 per cent. If the pain is severe the patient may appear collapsed, perspire freely, and have a retracted abdomen. There may be cough from reflex action. Apart from colic there is often gastro-intestinal disturbance, and gall-stones may be found in the feces. The patient may become excited.

(2) *Subjective Symptoms.*—In cases that are suffering from colic there may be nausea and digestive disturbance. If colic occurs there is pain and nausea, and often a feeling of much depression. The pain, which may be very intense, commences in the right hypochondrium, but may radiate to the back, umbilicus, left side, and even to greater distances. The pain may last only a few minutes or may persist for hours or days, and is liable to recur. There may be violent headache and sometimes there is marked dyspnea, due to a disturbed pulmonary circulation.

PHYSICAL FINDINGS.—*Observation.*—The appearance of the patient in hepatic colic is that of much suffering. The heavy perspiration, the collapsed look, and the rolling positions assumed by the sufferer suggests some acute intra-abdominal disturbance. Jaundice should be looked for and the retracted condition of the abdomen noted. The character of the vomiting should receive attention, as should also the state of the nervous system. The temperature may rise to 103° or 104° F. (39.5°-40° C.).

Palpation.—The gall-bladder is tender to pressure, and there is an area of tenderness at the ninth costal cartilage and extending some distance toward the navel. In more than one-third of all the cases the gall-bladder can be felt. There is usually a sensitive area at the level of the lower dorsal and upper lumbar vertebræ and extending from the posterior axillary border to the spine. The right rectus muscle is rigid. Calculi have been felt. The pulse is increased in frequency during an attack of gall-stone colic.

Percussion.—When the gall-bladder is distended the amount of enlargement can be made out by careful percussion.

Auscultation.—Stones in the gall-bladder have been made out in some cases by auscultation through a crepitation caused by percussion.

LABORATORY FINDINGS.—*The Urine.*—In those cases that suffer from chills and fever, or in which the gall-bladder becomes infected and inflamed, there may be albuminuria or glycosuria. In cases with jaundice the bile-coloring matter will be found in the urine and the stools will be light colored.

The Feces.—These should be thoroughly mixed with a large quantity of water and strained through a fine meshed sieve, or gauze bag. The solid portions are then examined for calculi.

SPECIAL EXAMINATIONS.—Exploration of the gall-bladder by a needle is not a safe procedure, and cannot be recommended, as some of its contents may escape into the peritoneum and give rise to peritonitis. An exploratory opening of the abdominal cavity with the object of examining the gall-bladder and large bile-ducts is a justifiable and proper course to follow, as it is safe and removes all doubt. The use of the *x*-rays and the screen is of very little value in the diagnosis of gall-stones, as they seldom throw a shadow, and, indeed, any shadow is likely to be obscured by the liver or other structures.

Diagnosis.—The essential consideration in diagnosis is the differentiation of hepatic colic from other forms of abdominal pain.

Gastralgia is usually relieved by vomiting, and may be traced to errors in diet. It is not often accompanied by vomiting, and there is not the same degree of collapse. In gastralgia the patient usually lies in the prone position, but in gall-stone colic the tendency is to roll about.

In gastric and duodenal ulcer there is the definite relation of the pain to the taking of food in these conditions. In gastric ulcer blood may be found in the vomit, and in the stools in duodenal ulcer. In both there is an absence of jaundice, distention of the gall-bladder, or calculi in the stools.

In lead colic there is obstinate constipation, the blue line on the gums, the presence sometimes of wrist-drop, and the history of an occupation that makes lead-poisoning a probability.

Renal colic is characterized by the direction of the pain down into the genitalia, the presence of blood or pus in the urine, and the passage of a calculus in the urine. There is also the absence of the signs due to biliary obstruction.

The crises of locomotor ataxia are accompanied by the other symptoms of the disease, such as the ataxia, the condition of the pupils, the loss of the knee-jerks, and the incoördination of movements.

Mucous colitis can be differentiated from other conditions by the nervous symptoms, and the occurrence of the mucous casts in the stools.

Appendicitis is found present in about 30 per cent of gall-stone cases, and gall-stones are found in about 10 per cent. of appendicitis cases. The four cardinal symptoms of appendicitis, namely, tenderness at McBurney's point, elevation of temperature, rigidity of the abdominal muscles, and vomiting, should prevent mistakes.

Acute pancreatitis is known by the sudden onset, the severe pain, the marked tenderness, persistent vomiting, profound collapse, epigastric distention, and the absence of indican, leucin and tyrosin from the urine.

Complications and Sequelæ.—THE GALL-BLADDER.—The distended gall-bladder or one containing many gall-stones may cause pressure on the duodenum, the pylorus, or common duct. The gall-bladder may rupture as the result of ulceration or inflammatory changes which may be of all grades from a mild catarrhal form to the acute phlegmonous and gangrenous types. Peritonitis and adhesions may result, and from infective processes there may be pylephlebitis. From pressure and adhesions there may ensue a great deal of indigestion and gastro-intestinal disturbance. Subphrenic abscesses may form.

THE CYSTIC DUCT.—Impaction of a calculus in the cystic duct can give rise to pressure upon the common duct, or a spasm of it, and in either of these ways cause jaundice. The cystic duct may become twisted, or obliterated.

THE COMMON DUCT.—When the calculus causes complete obstruction the jaundice is severe and permanent. In many cases the common duct is not completely occluded and some bile finds its way into the intestines and some infection finds its way into the ducts and gall-bladder, giving rise to intermittent attacks of fever. The spleen becomes enlarged. In at least 80 per cent. of these cases of calculous obstruction the gall-bladder is smaller than normal. The infection may ascend and cause abscess in the liver, there may be a pylephlebitis, and the bile-ducts may become stenosed by cicatricial tissue.

AMPULLA OF VATER.—A calculus in the ampulla may obstruct the flow of the pancreatic juice and its likelihood of reaching the intestinal canal. It may also cause bile to enter the pancreatic ducts, resulting in an acute pancreatitis. The obstruction of the pancreatic duct is a cause for the formation of a calculus in it, and the head of the pancreas may become fibrotic, or enlarged and indurated, closely resembling malignant disease. The pancreas may undergo degenerative changes and glycosuria may follow.

INTESTINAL OBSTRUCTION.—Calculi of sufficient size may enter the intestines and cause obstruction; or they may increase in size after passing into the bowel. As the result of inflammatory adhesions the gall-bladder or bile-ducts become adherent to the intestinal wall, and the calculi ulcerate their way through. The mortality in these cases is high, reaching, according to some statistics, as much as 50 per cent. The obstruction is in the ileum in nearly 70 per cent., in the duodenum in about 20 per cent., at the ileocecal valve in about 8 per cent., and in the sigmoid in 2 or 3 per cent.

COMMUNICATION WITH ADJOINING ORGANS.—The gall-bladder and bile-ducts may form communications with adjoining hollow organs. Thus perforations may occur into the duodenum, the colon, the pelvis of the kidney, a bronchial tube, or between the gall-bladder and the common duct. The opening made into the gall-bladder for the purpose of removing the gall-stones and drainage of the gall-bladder usually closes.

CANCER.—Nearly all cases of primary cancer of the gall-bladder and bile-ducts are due to the irritation and chronic inflammation originating from the presence of gall-stones. The percentage of malignant disease so caused does not fall below 80 per cent., and some place it at a higher figure.

Association with Other Diseases.—Cholelithiasis is sometimes associated with diabetes, renal calculi, defective conditions of metabolism, as gout, and with such gastro-intestinal disturbances as cause or result from cholelithiasis.

Clinical Varieties.—CALCULI IN THE GALL-BLADDER.—When the gall-stones remain in the gall-bladder in the large majority of cases—perhaps as high as 90 per cent.—they give rise to no symptoms. When symptoms do exist they are usually somewhat indefinite, as a feeling of fullness or weight in the region of the gall-bladder. There may be a dull pain radiating to the back, to the lumbar region, and to the right shoulder, and some feeling of nervous depression, headache, and epigastric discomfort. The gall-bladder may be enlarged in this variety, and a fremitus may be felt by the movement of several calculi against one another.

CALCULUS IN THE CYSTIC DUCT.—A calculus in the cystic duct may cause little disturbance, but the following conditions are liable to appear: The gall-bladder becomes distended with bile and mucus, and the latter may predominate to such an extent that the fluid becomes almost colorless. The contents of the gall-bladder may become purulent—an empyema of the viscus. The distended gall-bladder projects downward and is freely movable. It may attain very considerable size. If a number of calculi are present there may be crepitus on percussion. The distention may become great, or there may be ulceration, and rupture may result from either. Varying degrees of inflammation may result, from a mild catarrhal to a destructive phlegmonous type. Sometimes the gall-bladder atrophies.

CALCULUS IN THE COMMON DUCT.—When the obstruction is complete there will be severe jaundice. The history of pain may aid in differentiating this from the jaundice caused by pressure of a growth upon the common duct. When the obstruction is complete there is an absence of septic symptoms. Above the point of obstruction the ducts may become so distended as to be mistaken for the gall-bladder. If the obstruction is incomplete, in addition to the jaundice, the ducts above the gall-bladder become infected and there are septic phenomena, such as recurrent chills and fever. A suppurative process may extend into the gall-bladder and also as far as the intrahepatic bile-ducts. Sometimes there are distinct ague-like attacks without infection, the chill being severe and the temperature rising to 105° F. (40.5° C.). This is the recurrent hepatic fever of Charcot. In this variety of cholelithiasis the gall-bladder is usually contracted. As some bile finds its way into the intestinal canal urobilin is present in the urine, and when pancreatitis occurs the Cammidge test will reveal the characteristic crystals.

CALCULI IN THE BILIARY DUCTS.—In rare instances calculi may form in the biliary ducts within the liver. In a few cases these calculi have been very numerous, and have induced jaundice by the obstruction of a number of the intrahepatic ducts. There is an absence of biliary colic, but there is a hepatic cholangitis, and abscesses may form. The intrahepatic calculus may be large or branched.

Treatment.—PROPHYLAXIS.—The prevention of gall-stones is an important part of a medical practitioner's duties. People should be taught to take **proper and sufficient exercise. Deep breathing** is a valuable means of promoting the flow of the bile. The **abusive use of alcohol should be forbidden,** and all **errors of digestion corrected.** The **free consumption of water** increases the flow of bile and does much to prevent infection and inflammation of the gall-bladder and bile-passages. There is no disinfectant of these organs equal to a plentiful flow of normal bile. In all cases where the biliary passages or gall-bladder are suspected of being congested, inflamed, or infected, **sodium salicylate** in doses of grains 15 (1 gram), or **hexamethylenamin,** grains 5-10 (0.325-0.650 gram), thrice daily, will prove valuable. Constipation must receive attention and some form of saline meets the requirements best. Perhaps there is none better than **sodium phosphate** in doses of 4-8 drams (15.5-31 grams). As a corrective of a catarrhal condition of the biliary apparatus, sodium phosphate in dram (3.90 gram) doses several times a day is very useful. All cases of gastro-intestinal disturbance should be given proper treatment, and every case of typhoid fever ought to be kept under observation for some time. It would be well, as a routine, to give these cases a course of treatment with the drugs just mentioned, as the best means of preventing or correcting infection of the gall-bladder.

GENERAL MANAGEMENT.—The general treatment of gall-stone cases consists in **suitable exercise,** such as walking, rowing, saddle-riding, light gymnastic training, **deep breathing, and the wearing of loose and warm clothing.**

Diet.—The ingestion of food favors the flow of bile; consequently the subjects of gall-stones should take a small meal often. It is never well to eat too freely, for fear of causing gastro-intestinal disorders. Starchy foods should be taken in moderation and bread is better toasted. Sugars should be limited. It is generally conceded that fats should also be considerably curtailed. On the other hand, most vegetables and protein foods may be indulged in rather freely. It has been definitely settled that fatty foods have no power to dissolve biliary calculi, or to cause their expulsion; and, as they may give rise to indigestion, they should be used with due caution. A cholesterol-free diet has been advised, and for this purpose yolk of eggs, peas, fatty meats, and fried foods should be excluded. Alcoholic stimulants are best avoided, and especially ale, stout, porter, champagne, beer, and liqueurs. If any are taken, they had better be light wines and a little whisky and water. Patients with gall-stones should be encouraged to drink a good deal of water, more particularly before meals, and hot water has enjoyed a good deal of favor. Many saline spring waters are useful, as they increase the flow of bile and prevent the formation of calculi, help to wash away those that are formed, and assist in relieving the catarrhal condition of the mucous membrane. The waters from Carlsbad, Harrogate, and Hot Springs may be tried. They should be drunk hot, in large quantities, and two or three times a day, before ingestion. They act best when sipped slowly.

Local Treatment.—M. E. Rehfuss has recently recommended local treatment by the passage of the **duodenal tube.** While bile comes up, the tube is connected with the Murphy drip apparatus, and the solution is run in at the rate of about 60 drops (4 c.c.) per minute. From 4 to 6 ounces (118.4 to 178 c.c.) of solution containing **argyrol, protargol, methylene blue, or salicylic acid** are used. **Urotropin (hexamethylenamin)** may be introduced into the duodenum in doses of grains 60 (4 grams). Concentrated solutions of **sodium bicarbonate, sodium sulphate or phosphate** have been found helpful. Careful but thorough **colon irrigation** should receive attention. The tube should be of such construction as to permit both an in- and an out-flow.

Massage.—Massage in the treatment of gall-stones is condemned by most writers. The author has found signal benefit from it when carefully employed. Firm massage over the gall-bladder and bile-ducts must be condemned; but a gentle massaging of the entire abdomen stimulates intestinal peristalsis, increases the flow of bile, aids in the passage of calculi, and prevents further formation.

Treatment of Symptoms.—*The jaundice* accompanying biliary colic is treated by ordering a **light, digestible diet,** by the use of **saline laxatives** to keep the bowels acting, and the prescribing of **urotropin (hexamethylenamin),** grains 5 (0.325 gram), combined with grains 10 (0.65 gram) of **sodium salicylate,** every four hours. *Itching* may be relieved by **calcium lactate,** grains 15 (1 gram), several times daily. (For further information on the treatment of these symptoms *see* the section on Jaundice.)

The pain of biliary colic may be relieved by the local application of heat in the form of **fomentations, poultices, or a rubber bag containing hot water.** Medicinally **antipyrin, aspirin, phenacetin, and belladonna** may be tried for the pain. These, however, are not at all reliable, and it is usually necessary to administer **morphia and atropin** in combination—grain ¼ of the former and grain 1/100 of the latter—hypodermically. In some cases the pain may be so violent as to demand anesthesia by ether or chloroform. **Methyl salicylate,** 1 or 2 drams (3.90 or 7.80 grams), painted over the liver and covered with gutta percha is a good local anodyne. Some have obtained benefit from **turpentine,** minims 15 (0.92 c.c.), spirits of **ether,** minims 30 (1.90 c.c.), and **chloroform water,** one ounce (31 c.c.). One large dose of salicylate of soda at the beginning of the attack, grains 30 to 45 (1.95 to 2.92 grams), has found favor with some. A **hot bath** sometimes affords much relief.

The vomiting during an attack of biliary colic may be assuaged by

draughts of hot water containing **bicarbonate of soda,** 1 dram (3.90 grams), to the pint of hot water. **Bismuth, dilute hydrocyanic acid** and **iced effervescent waters** may also be given a trial. **Heat over the stomach** and **iced champagne** have been found very useful.

SPECIFIC TREATMENT.—The only specific treatment that has been found of any use is the employment of an **autogenous vaccine of the colon bacillus** obtained from the feces of the patient, and proved to be the infecting agent by the agglutination test. Pus foci must be searched for, and, if found, cultures made from these, so as to secure a polyvalent vaccine. Of the colon bacillus, 25,000,000, and of the pus organisms, 100,000,000 are employed as the initial dose. The writer has derived benefit from this treatment in 2 cases, operation being refused in one, and contra-indicated in the other.

MEDICINAL TREATMENT.—Much can be done for patients suffering from cholelithiasis by the judicious selection of drugs. It must be laid down as an axiom that the best agent known for the prevention of gall-stones and their solution or passage, if already formed, is **an abundant flow of normal bile.** For the mucous membrane of the gall-bladder and the bile-ducts it is nature's solvent, antiseptic, and correctant. It must be the physician's aim, therefore, to promote a flow of normal bile. For the purpose of curing such patients, **olive oil** has been lauded by many, but, unfortunately, *there is no ground for the belief.* Olive oil cannot enter the biliary capillaries in the liver and descend through the hepatic ducts, nor enter the gall-bladder, nor pass down the common duct. Nor can it ascend through the common duct to the gall-bladder. If a gall-stone lay in the intestinal canal it might aid in its onward movement.

The number of drugs that can increase the flow of bile, true cholagogues, are very few. **Ipecacuanha** is one of them, and may be given in doses of grains 2 (0.13 gram) four times a day. **Sodium phosphate** in dram (3.90 grams) doses several times daily is worthy of a trial. Bile is thought well of by some, and is best given in the form of the **salts of the bile acids. Colanin** is another preparation from bile. They have no very marked influence on the flow of the bile, but preparations of bile may aid in relieving accompanying indigestion. **Salicylacetic acid (aspirin)** has some merit as a cholagogue, but cannot be recommended as highly as others. The best known stimulus to the flow of bile is **sodium salicylate,** which may be given in doses of grains 10-15 (0.65-1 gram) every four hours. To these doses **sodium benzoate,** grains 5 (0.325 gram), may be added. An excellent combination is **sodium salicylate and urotropin,** about grains 15 (1 gram) and grains 5 (0.325 gram), respectively, four times a day. The urotropin (hexamethylenamin) is excreted freely in the bile, and is a very valuable biliary antiseptic. **Durande's mixture,** consisting of ether (4 parts) and turpentine (1 part), has had considerable reputation both as a cholagogue and as a solvent of gall-stones. It is really neither, and only acts as an antispasmodic. **The bowels should be kept loose by means of saline aperients, but drastic purgatives must be avoided.**

SURGICAL INDICATIONS.—One of the responsibilities resting upon the medical attendant is that of deciding when a case of cholelithiasis calls for surgical treatment. The following principles may be laid down as indicating the need for operation:

1. In all cases where the attacks of jaundice, colic, and fever become more frequent and severe, and do not yield to medical treatment. These cases drift into chronic invalidism or fatal cholemia.
2. In cases where a calculus is impacted in the cystic duct and the gall-bladder is permanently distended with mucus or pus.
3. In all cases with the history of gall-stones, when the symptoms of perforation and peritonitis occur.
4. When acute intestinal obstruction comes on in a person with the history of biliary colic, jaundice, and fever.
5. When severe gastric symptoms occur in one who has had hepatic colic, and there are reasons to suspect adhesions between the gall-bladder and the stomach or pylorus.
6. In cases of fistulæ between the gall-bladder and other viscera, or to the outside of the body.
7. If the symptoms of pain, fever, chills and jaundice point to a severe infective inflammation of the gall-bladder or bile-ducts, the gall-bladder should be drained.
8. When jaundice in a patient with the history of biliary calculi becomes chronic, an operation should be performed before the condition of cholemia and exhaustion becomes too advanced. The period the patient is under observation may vary from a few weeks to several months.
9. Cases with indications of severe peritonitis in the region of the gall-bladder.
10. Generally speaking, gall-stones should be removed while they are in the gall-bladder. This would avoid impaction in the ducts, the occurrence of troublesome cholecystitis, perforation and cancer.
11. Cases of suppuration of the gall-bladder, the bile-ducts, the liver, or in the immediate vicinity demand surgical treatment.

MANAGEMENT OF CONVALESCENCE.—During convalescence the aim of the medical attendant should be to secure the best possible condition of health in the digestive organs. The diet must be carefully regulated, and all excesses in food and stimulants prohibited. Proper exercise is very desirable, improperly fitting corsets and tight lacing being avoided. The treatment for cholecystitis should be maintained for such a time as is likely to avert the recurrence of calculi; and the axiom should ever be remembered: "A healthy gall-bladder and a normal flow of bile and no gall-stones."

Prognosis.—AS TO RECOVERY.—Very many cases of gall-stones are latent, and the diagnosis is only made when a postmortem is performed. This amply proves that the presence of calculi in the gall-bladder is not very serious. When symptoms of their presence, however, occur, under judicious medical and surgical treatment, the recovery rate should be high. Now that gall-stones is recognized as a surgical condition, the death rate has been very greatly reduced. The main danger is that of allowing patients to drift on until the biliary passages are seriously diseased, or until the health of the patient is much impaired before surgical relief is afforded. With the efficient state of gall-bladder surgery at the present day, there is no justification for such a course. When gall-stones migrate into the ducts the subsequent history of the patient changes rapidly for the worse, as compared with cases where they remain in the gall-bladder. Symptoms of obstruction now come on, jaundice occurs, infection of the ducts and gall-bladder adds to the dangers of the case, and abscesses and perforations may result. In cases with obstructive symptoms unrelieved, the death rate is high. The formation of adhesions renders prognosis less favorable. If the patient passes faceted calculi in the stools it may be presumed that other attacks will occur, whereas the discharge of an unfaceted calculus is ground for the belief that there may be no others.

Mode of Death.—The mode of death may be by exhaustion and the condition of cholemia. Death may result from infection and the formation of abscesses. The occurrence of peritonitis from infection or perforation may lead to a fatal termination. Pancreatitis may also be induced, which increases the gravity of the situation. When a calculus escapes into the intestines and gives rise to intestinal obstruction, the mortality in recorded cases, not relieved by operation, is about 50 per cent. Death may occur during an attack of hepatic colic through heart-failure caused by the severity of the pain. Death may also be due to the condition of the pancreas, 50 per cent. of the cases of acute pancreatis and 75 per cent. of the chronic type being caused by gall-stones or the accompanying infection. In cholecystostomy operations the death-rate is about 1 per cent., and in cholecystectomy it is about 2 per cent.

Pathology.—The pathological processes found in connection with gall-stones are those found in chronic infective inflammation of the gall-bladder and bile-passages. They have been fully described in the sections on Cholecystitis and Cholangitis. This inflammation adds cholesterin, bacterial cultures, and calcium exudate to the production of mucus. These gather into masses in the formation of calculi. With these are found bile-pigments, epithelium and bile-salts. Some calculi are composed mainly of bilirubin-calcium, and others principally of cholesterin. It has been observed that a clump of bacteria may form the nucleus around which other constituents gather. It has been proven that cholesterin is a product of the mucous membrane, and the amount is increased when it is inflamed. The bilirubin-calcium is the chief cement binding the various constituents together.

Some calculi are almost entirely formed of cholesterin, while others are composed of bile coloring matter. Both varieties are soft and usually small. Some calculi are laminated and formed by cholesterin and calcium or bilirubin-calcium, being deposited in layers. The mixed cholesterin and bilirubin-calcium calculi are the most common and very much in size even to one large enough to completely fill the gall-bladder. Calculi composed only of bilirubin-calcium are usually small, ranging from that of sand to a pea. The more cholesterin and coloring matter the softer, and the more calcium salt the harder the calculi. In color calculi vary greatly. Those composed mainly of cholesterin are whitish, yellow, or brownish, or greenish, according to the admixture of coloring matter. As the bilirubin and biliverdin increase in amount, they tend to become brownish or greenish. Calculi formed of calcium carbonate are rare and give a shadow with the *x*-ray, while others do not. The site of their formation is usually the gall-bladder, but calculi have formed in the bile-ducts. When the intrahepatic ducts have become infected, calculi have formed within the liver. Calculi may vary greatly in shape, being round, oblong, faceted, smooth, rough, or even branched, or a conglomeration of a number in a common covering.

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HYDATIDS OF THE LIVER

(Echinococcus Cysts of the Liver)

Etiology, p. 196—Symptomatology, p. 196—Diagnosis, p. 197—Complications, p. 198—Clinical varieties, p. 199—Treatment, p. 199:—Prophylaxis, p. 199—Prognosis, p. 200—Pathology, p. 200—Historical summary, p. 200.

Etiology.—PREDISPOSING CAUSES.—*Climate.*—The disease is more common in some districts than in others. It is more frequently met with in Iceland than in England, and in London than in the suburbs. In Australia it is most common in the bush districts.

Sex.—Males suffer slightly oftener than females, perhaps about in the ratio of 3 to 2. In Iceland, however, women are twice as often affected as men.

Age.—The disease increases in frequency up to fifty years of age and thereafter steadily declines.

Race.—People who live in close companionship with their dogs are specially liable to become the victims of hydatids.

EXCITING CAUSE.—A mature *Tænia echinococcus* in the dog is about one-sixth inch (4 mm.) long, and consists of a head and three sections. The last contains about 500 ova. When this section separates it is discharged in the feces and the ova are then set free. These may be taken into the alimentary canal on food or in water. The ova then develop into embryos which are carried by the portal veins to the liver.

Symptomatology.—CLINICAL HISTORY.—*Mode of Onset.*—The development of the cyst and the progress of the disease may be very slow, and not revealed by any active symptoms. In at least half the cases the disease is latent, and may only be discovered by an examination for some other purpose, or at a postmortem examination.

Symptoms during Progress of Disease.—(*a*) *Objective Symptoms.*—When the cyst or cysts attain considerable size there may be a visible enlargement or prominence in the region of the liver, and the lower ribs may be everted. There may be hypertrophy of the left lobe, which causes a fullness on that side also.

(*b*) *Subjective Symptoms.*—As the cyst enlarges there is a feeling of fullness, and very rarely pain and tenderness are experienced. There may be dyspnea due to pressure.

PHYSICAL FINDINGS.—*Observation.*—Tumor in the hepatic area becomes more and more pronounced with the growth of the cyst or cysts. The ribs are bulged outward. Jaundice, ascites, a caput medusæ, and edema of the legs may be met with in some cases, owing to the pressure exerted by the cyst.

Palpation.—Palpation may elicit some tenderness, and the outline of the tumor can in many cases be made out. The shape varies with the size and location of the cyst, but the enlargement is usually smooth, tense and elastic.

Percussion.—The area of liver dulness is increased and modified in shape. It may extend upward, simulating a pleuritic effusion. Percussion over the swelling may discover a thrill. This was thought at one time to be very positive evidence of hydatid cysts, but it is not a very reliable sign, as it may be absent, or the thrill may be due to other cysts, to a hydronephrosis, or to an encysted ascites.

SPECIAL TESTS.—If some of the fluid is withdrawn from the cyst, the presence of the hooklets, the membrane of the cyst, and the scolices may be discovered. The fluid is neutral in reaction, slightly opalescent, of low specific gravity (1.002-1.015), contains mucin but no albumin, some inosite, sugar, leucin, traces of cholesterin, and a small quantity of chlorid of sodium. When the parasites die and the cyst becomes sterile the fluid will contain albumin and become toxic. The presence of glycogen is regarded as evidence of activity in the cyst. It is, however, a dangerous practice to puncture the cyst for fluid, and should not be resorted to as a means of clearing up the diagnosis. These elements may be discharged into the intestinal canal or a bronchial tube by the rupture of the cyst.

Diagnosis.—CLINICAL HISTORY.—The clinical history is often indefinite and the progress of the disease slow. The signs may become pronounced, while the symptoms attract but little attention. As the case goes on the tumor may become large, and the pressure may cause jaundice and pain. In a few cases perihepatitis has occurred. The patient may experience a feeling of weight and fullness in the region of the liver. Cases may last for many years.

PHYSICAL FINDINGS.—There is the presence of a smooth, round tumor, tense and fluctuating. A thrill may be detected. These features, coupled with the history of slow growth, the slight amount or absence of pain, and the freedom from constitutional disturbance, would justify the diagnosis of a hydatid cyst.

LABORATORY FINDINGS.—The laboratory findings are those already stated, namely, the discovery of the hooklets, parts of the cyst membrane, and the characteristic fluid. These may be obtained by aspiration, or when the cyst ruptures into the intestinal canal or into a bronchial tube.

DIFFERENTIAL DIAGNOSIS.—*Abscess.*—Abscess in the liver is accompanied by the history of sepsis, or dysentery, or pylephlebitis. The hydatid cyst may suppurate, however, when the condition becomes that of a hepatic abscess.

Cancer.—Cancer of the liver is much more rapid than is that of a hydatid cyst. There is emaciation, pain, and a marked cachexia. There is also a greater tendency to jaundice, and the tumor is irregular and devoid of fluctuation. There is no thrill.

Syphilis.—When syphilis affects the liver the enlargement is hard; there may also be albuminuria. A history of syphilis can usually be made out, and the blood test would assist in making a diagnosis. The result of specific treatment is an important feature.

Distended Gall-bladder.—When the gall-bladder is distended the tumor is pear-shaped, and corresponds with the end of the ninth costal cartilage. The tumor will move laterally without moving the liver, whereas a hydatid cyst is movable only with the liver. Further, there will be a history of previous local disease, such as cholecystitis, gallstones, or some obstruction of the bile-ducts.

Lardaceous Degeneration.—Enlargement of the liver due to lardaceous degeneration, or as a feature of leukemia and cardiac disease, is distinguishable from enlargement caused by a hydatid cyst by the clinical history of these affections.

Pleurisy with Effusion.—When a hydatid cyst extends upward the condition may closely simulate a pleurisy with effusion. An *x*-ray plate may reveal the outline of the tumor, and the aspiration of a portion of the fluid should enable one to clear up any doubt. Expectorated matter may contain the hooklets or sheds of the cyst. The development of a pleuritic effusion is more rapid than the hydatid cyst.

Kidney Tumor.—Tumors connected with the kidney evert the ribs less and encroach upon the thorax less than does a hydatid cyst. Then the renal tumor is covered by the colon, and there is a line of separation between the tumor and the liver. The kidney tumor does not move with respiration. A hydronephrosis may disappear with diuresis.

Pancreatic Cyst.—Pancreatic cysts or cysts around the pancreas are behind the stomach, whereas the hydatid of the liver is in front of it. They are more deeply located, also, and are separated from the liver by a zone of resonance. A differential diagnosis can be made by inflating the stomach with air.

Simple Cyst.—Simple cyst of the liver may give rise to some doubt. There are usually evidences of a cystic condition in other organs, as the kidney. There are also frequently associated with simple cysts certain malformations, such as harelip, spina bifida, hypospadias, and other abnormalities.

Complications.—RUPTURE.—(*a*) The cyst may rupture into the peritoneum, causing sudden collapse and severe peritonitis. On the other hand, the rupture may cause very little in the way of acute symptoms, but cause a hydatid infection of the abdominal cavity—a condition that has been spoken of as pseudo-tuberculosis.

(*b*) The rupture may be into the pleural cavity. This will simulate a pleurisy with effusion. The fluid may become purulent, resulting in an empyema.

(*c*) The cyst has also ruptured into the lung—usually the right—causing the expectoration of the contents of the cyst, and sometimes giving rise to hemorrhage, and extreme dyspnea.

(*d*) Sometimes the cyst may open into the gall-bladder or bile-ducts. In such cases there may be no symptoms, or the case may resemble biliary colic. The contents of the cyst may thus escape and a cure result. The cavity of the cyst may become infected from the bile-passages, and suppuration ensue.

(*e*) Rupture into the pericardium, stomach, and intestines is rare. Rupture into the vena cava has been known to occur.

SUPPURATION.—A hydatid cyst may suppurate, when it has a strong tendency to rupture and spread the infection. The infection may reach the cyst cavity from the lungs, the pleuræ, the bile-passages, the intestines, or from aspiration. The organisms found have been the colon bacillus, streptococci, several strains of staphylococci, and *Bacillus aërogenes capsulatus.* Old and sterile cysts have been known to suppurate —one of the late dangers of hydatids. Hemorrhages may occur.

Clinical Varieties.—The clinical varieties of liver hydatids are dependent upon their number, which may be only one, or several; upon the size of the cyst, which may be small, or almost fill the abdomen; upon the speed of growth, which may be very slow, occupying many years, or very rapid; and upon the location of the cyst, which may be deep in the liver substance, or projecting downward into the abdomen, or upward toward the chest, or forward against the parietes.

Treatment.—PROPHYLAXIS.—The ova are taken into the digestive canal on food and in water. It is well, therefore, that all vegetable foods should be properly washed before being eaten, and that the water supply should be protected against contamination by dog excrement. Those who keep dogs should take proper precautions along these lines. In localities where the disease is known to exist strict care should be exercised with regard to raw vegetable food and drinking water. The offal from the slaughter of animals should be destroyed to prevent the infection of dogs. Stray dogs should be destroyed.

GENERAL MANAGEMENT.—*Trocar and Aspirator.*—The earliest method of treatment consisted in **tapping the cyst** and allowing the fluid to run off. To insure complete removal of the fluid, an aspirator may be attached to the trocar. This plan has often succeeded, but is accompanied by many risks, such as peritonitis, the infection of the cyst cavity, or the death of the patient by intoxication from products liberated from the dead parasites.

Injections.—Some have treated hydatids by injecting some antiseptic agent into the cyst, for the purpose of destroying the echinococci. Carbolic acid, iodin, alcohol, formalin and bichlorid of mercury have been used. This method of treatment is dangerous and should not be adopted.

SURGICAL TREATMENT.—*Incision and Drainage.*—In this operation an opening is made through the abdominal walls and the cyst exposed. The abdominal cavity is carefully protected by gauze packs. The cyst is drained and brought well up into the opening. An incision is made into it through which the index finger is inserted, by which daughter cysts and the endocysts are broken up. The cavity is flushed with normal saline solution and then packed with gauze. The liver is now stitched to the abdominal walls, the gauze removed and a large drainage tube is passed well into the cavity.

Enucleation.—This operation is conducted in the same way as for drainage, but when the cyst has been emptied and brought up into the opening, it is incised and the endocyst is carefully removed by the finger, sponging and flushing. The cavity is then obliterated by bringing the ectocyst into apposition by catgut sutures. When the cavity has been completely suppressed, the wound in the liver is closed by catgut, the liver is then replaced in the abdominal cavity, and the external opening closed. If drainage must be used, rubber tissue is the best.

Prognosis.—AS TO RECOVERY.—In the case of small cysts, the patient may recover through the death of the parasite; but such as have undergone this spontaneous cure have sometimes become infected and suppurated. If the cyst is large the prognosis depends upon the results of surgical treatment.

Mode of Death.—The mode of death is threefold. (*a*) The parasites may die spontaneously or as the result of aspiration, and liberate such an amount of toxic albuminous products as to prove fatal. (*b*) The cyst may suppurate and rupture, causing serious sepsis. (*c*) Or it may rupture into the peritoneal cavity or some adjacent organ. When it takes place into the peritoneal cavity the death-rate is 90 per cent., into the pleura 80 per cent., into the bile-ducts 70 per cent., into the bronchi 57 per cent., stomach 40 per cent., intestines 15 per cent., and externally 3 per cent. These dangers are averted by proper surgical treatment.

AS TO FUNCTION.—Prognosis as to function is satisfactory in most cases of spontaneous cure. In cases that are treated surgically the liver acts normally. In those cases where the cyst communicates with bile-ducts and bilary fistulæ result, there may be serious impairment to health. Cholecystitis and cholangitis, through infection from a suppurating cyst, may result in impairment of hepatic function.

Pathology.—At operation or postmortem the cyst is found to consist of two layers. The outer layer is laminated and composed of elastic substance, and is called the ectocyst or cuticle. The inner is the endocyst, and is known as the germinal or parenchymatous layer, and is granular. The cyst contains fluid in which may be found scolices and hooklets. From the parent cyst daughter cysts may develop, on the surface of which scolices may be found having four suckers and a circle of hooklets. These daughter cysts become separated from the parent cyst and lie free in its cavity. These daughter cysts may form buds that evolve into granddaughter cysts. Thus the endocyst forms brood capsules that become daughter cysts, and these again form brood capsules that become granddaughter cysts. From the endocyst arise the buds that form scolices, which in another host—the dog—become the *Tæniæ echinococci.* A scolex is 0.3 millimeter long and is composed of the head and one section. In the fully grown tapeworm this latter, by transverse division, becomes three sections. The hooklets measure 0.04 milimeter. Surrounding the entire cyst, from irritation of the liver, a fibrous capsule is formed.

Historical Summary.—The disease has been known since the time of Hippocrates. He devised the operation of tapping the cyst. In 1871, Fagge and Durham employed electrolysis. In 1890, Baccelli recommended injecting an antiseptic into the cyst. In 1883, Knowsley Thornton enucleated the sac.

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ACTINOMYCOSIS

(Ray-fungus)

Etiology, p. 201—Symptomatology, p. 201—Diagnosis, p. 202—Treatment, p. 202—Prognosis, p. 202—Pathology, p. 202—Geographical distribution, p. 203.

Etiology.—Predisposing Causes.—*Age.*—The disease occurs most frequently between the ages of twenty and forty-five years.

Sex.—In regard to sex, the ratio is three males to one female.

Exciting Cause—The Organism.—The organism of the disease is *Actinomyces bovis*, found in the alimentary canal of cattle. The small clumps produced by the parasite were first noted by Langenbeck, in 1845. In 1877, Bollinger discovered the fungus in the ox; and Israel found it in man, in 1878. The organism was named actinomyces by Hartz. It is held by some to be taken into the body on cereal grains, but this is denied by others.

General Characteristics.—Fungus is a streptothrix and assumes the forms of filaments, clubs, and cocci.

Mode of Conveyance.—The fungus is conveyed to man through food, drink, or by contact with an actinomycotic sore. Generally, man is infected by way of the mouth, but it may take place through a wound in the skin. The ray-fungus may spread to other organs, by extension, or may become metastatic through the blood or lymph-channels, in the leukocytes. The primary seat of infection may be the lung—by aspiration—the mouth, throat, or the vagina.

Symptomatology.—In the early period of the disease the symptoms may refer to abdominal trouble, such as appendicitis; and there may be pain, constipation, and a tumor somewhere in the abdomen. Later on when the liver becomes affected the symptoms may resemble empyema, or an abscess in the abdominal wall, around the kidney, or beneath the diaphragm. When the liver is enlarged and fever and pain are present the resemblance to hepatic abscess is close. Jaundice and ascites are very rare. Anemia and leukocytosis are well marked. At first the progress is slow and resembles a tuberculous infection.

Diagnosis.—What may be suspected to be a case of actinomycosis is only made certain by finding fungus in pus from the liver or some other part of the body. Until the fungus was discovered the condition was mistaken for sarcoma, suppurating hydatid, hepatic and perinephritic abscess, gumma of the liver, suppurative cholangitis, pulmonary tuberculosis, and empyema. Actinomycosis reacts to the tuberculin test, and so an error may be made, in mistaking the case for tuberculosis. The pus contains the characteristic sulphur-like granules.

Treatment.—Medicinal Treatment.—The chief drug is **iodid of potash.** This should be given in large doses, 2 or 3 drams (8 or 12 grams) daily.

Local Treatment.—From 15 to 30 minims (1 to 2 c.c.) of a one per cent. solution of potassium iodid in water, may be injected into the tumor every third day; *x*-**rays** have proven useful.

Specific Therapy.—A **vaccine** prepared from the fungus has effected a cure. **Tuberculin** has also been employed with benefit, and a **protein** from the culture of the *Staphylococcus pyogenes aureus* has been of use.

Surgical Indications.—If the abscess is opened it should be thoroughly cleaned out, and diseased tissue curetted away. The wound should be well disinfected, and care taken to avoid infection of any other part. **Sulphate of copper**, grain 1 (0.065 gram), three times a day, **and irrigation with a 1 per cent. solution of the same** have been advised.

Prognosis.—In actinomycosis of the liver the prognosis is very unfavorable. The disease is seldom recognized in its early stage when treatment might be effective. In the later stage the infected area becomes infected with pyogenic organisms and there is considerable necrosis of tissue—circumstances that add much to the gravity of the case. The death-rate is very high.

Pathology.—The liver is enlarged, especially at the location of the actinomycotic infection. The abscess is made up of numerous small cavities coalescing in a honey-comb fashion. The abscess resembles a sponge saturated with pus. There is usually only one abscess, but sometimes there are several. The mixed infection gives rise to pyemic features and the possibility of metastasis. The abscesses vary in size from that of a millet seed upward. The abscess may rupture into the abdominal cavity, into the lung, into the stomach, or beneath the diaphragm. Sometimes the liver becomes adherent to the abdominal wall and the abscess opens externally. There is a process of fibrosis and pigmentation in the liver around the abscess. There is a tendency to atrophy of the liver-cells, and the organ may show fatty or lardaceous changes. The ray-fungus may cause suppuration without the presence of other organisms. The early stage of the disease closely resembles tuberculosis in the formation of painless, granulation tissue. As the tumor enlarges there is an active proliferation of the surrounding connective tissue, and gradually the center of the mass softens.

Geographical Distribution.—The disease is rare in America and Britain, but more frequent in central and eastern Europe. Fully 50 per cent. of all the cases affect the head and neck; 20 per cent are in the digestive organs, and about 15 per cent. are met with in the lungs. Occasionally the disease begins in the skin.

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CYSTIC DISEASE

Etiology, p. 203—Symptomatology, p. 204—Diagnosis, p. 204—Treatment, p. 204—Prognosis, p. 204—Pathology, p. 204.

Etiology.—PREDISPOSING CAUSES.—*Age.*—Some cases have been observed to occur in infancy; and it is the opinion of many pathologists that the cases occurring in adult life are really cases that were congenital but have been latent during a great part of life. Multilocular cysts in the adult usually appear after fifty years of age.

Sex.—Men and women suffer in the ratio of 3 to 1.

THEORIES AS TO ORIGIN.—Some have taught that cysts are inflammatory in origin, and begin as an intrahepatic pericholangitis. Others regard the process as a degenerative one—a certain number of the liver-cells having disappeared, the area becomes surrounded by a fibrous wall. Some regard the cysts as outgrowths from the bile-ducts, or dilated new bile-ducts. The theory has also been advanced that some of the hypoblastic cells develop irregularly and form cystic areas. The two most acceptable views are the inflammatory origin and the development from the hypoblastic cells.

Symptomatology.—Enlargement of the liver may be surmised to be caused by the presence of cysts when a similar cystic condition is discovered in the kidneys. The cyst or cysts may be small and give rise to little or no disturbance, or they may be so large as to fill the entire abdomen. They may be mistaken for hydatid cysts, ovarian cysts, or a dilated gall-bladder. The cystic condition of the kidneys, which is almost invariably present, can generally be recognized. The patient may present symptoms resembling uremia; albumin is sometimes found in the urine.

Diagnosis.—The diagnosis rests upon the discovery of hepatic enlargement, and the condition of cystic kidney. Care must be exercised to differentiate between this condition of the liver and a distended gall-bladder, hydatid cysts of the liver, ovarian cysts, and hydronephrosis. A study of the clinical course of these conditions will enable one to arrive at a correct conclusion. Cysts in the liver are generally a part of the existence of cysts elsewhere, as in the ovaries, the tubes and the kidneys. Other deformities are frequently present, such as spina bifida, webbed fingers, etc.

Treatment.—The treatment is general and expectant. An effort should be made to avoid the occurrence of uremia. If the cyst becomes large it may be emptied and the cyst-wall removed by careful dissection and sponging. The cavity is closed by catgut sutures. A few cases have been successfully treated in this manner.

Prognosis.—When the cystic condition becomes manifest early in infancy, or is marked at the time of birth, the prognosis is decidedly unfavorable. When the cysts do not appear until adult life the prognosis is much better and may admit of successful removal. In cases coming on in adult life the cyst may increase to such a size as to produce very troublesome pressure symptoms and threaten life. In some cases of congenital cyst, the patient may survive and the cyst only become large later in life. In this way most of the adult cases are explained.

Pathology.—In most of the infantile cases the liver is but little enlarged, if at all. In a rare case, however, the enlargement is extreme. In most cases the cysts are very small and almost invisible to the naked eye. A few small cysts may appear on the surface of the liver. The portal spaces are fibrosed and in them some cystic dilatations. The larger biliary ducts appear normal. The cysts contain a clear fluid. In the adult the cysts tend to merge and form a lesser number, but of larger size than in the infant. Usually only a few, or perhaps only one, of the cysts becomes enlarged. They are usually of the size of a walnut, though some have contained one or two pints, and even filled the abdomen. On section the cyst-wall is found to be composed of fibrous tissue. The liver is more or less completely permeated by the cavities. The contents of the cysts is usually a clear fluid, but it may be darkish from hemorrhage, and is sometimes thick and colloid. The fluid contains calcium oxalate, leucin, protein, creatinin, cholesterin, urea, chlorids and epithelial débris, but not bile. There is a general tendency toward fibrosis throughout the liver. In the portal spaces numerous tubules are found, lined with epithelium, surrounding the interlobular veins. The cysts arise in the portal spaces in connection with these epithelial extensions.

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BENIGN TUMORS

Adenoma, p. 205: Symptomatology, p. 205—Diagnosis, p. 205—Clinical varieties, p. 205—Treatment, p. 206.
Fibroma, p. 206.
Angioma, p. 206: Symptomatology, p. 206—Diagnosis, p. 206—Treatment and prognosis, p. 206.

Adenoma.—Symptomatology.—The symptoms in adenomata of the liver will vary with the variety, number and size of the new formations. As a rule, these tumors do not attract much attention. Sometimes cystic changes may occur and the enlargement become apparent. In rare cases jaundice has been caused by pressure. If the adenoma is the result of adrenal inclusion the tumor may become malignant. Multiple adenomata are frequently associated with cirrhosis, when the symptoms of the latter condition will be present.

Diagnosis.—The diagnosis in most cases must remain in doubt until an exploratory opening is made. There may be discoverable enlargement, or jaundice, or ascites, but the nature of the new growth can rarely be determined without the performance of a laparotomy. Usually there are few or no symptoms, unless an adenoma develops into a cyst of some size, or the adrenal variety becomes malignant.

Clinical Varieties.—The varieties are etiological and histological.

1. One kind of adenoma is merely a mass of liver-cells enclosed in a fibrous capsule. They are usually solitary, though several may be found. Though these tumors are generally small, occasionally they may become large and measure as much as nine inches in diameter. The growth may become congested, with a tendency to cellular necrosis. In

time the cells become modified in shape, and the structure resembles a tubercle. The liver-cells may be so arranged as to form tubules lined with epithelium.

2. The second variety is a tumor formed from the intrahepatic bile-ducts. These growths crowd in upon the liver substance but do not become part of it. They are tubular adenomata lined with columnar epithelium. The mucous glands in the biliary tubes may take part in their formation. Occasionally such a tumor may become cystic and enlarge. This variety may be solitary or multiple.

3. The suprarenal gland variety is the result of inclusion of some adrenal cells in the liver. These become encapsuled and may remain dormant. They have been known to become malignant.

4. Adenoma hyperplasticum is that form in which there is a hyperplasia of the liver-cells to compensate for cell atrophy, the result of cirrhosis, malaria, chronic venous engorgement, or gradual atrophy of the liver-cells from certain food poisons, such as the mussel. In this variety of adenoma there are many nodules in the liver, and they are merely an exaggeration of the hobnails observed in portal cirrhosis. They may undergo fatty and necrotic changes, or become white and resemble caseous tubercles. The necrotic material may cause thrombosis of the hepatic veins. Some have regarded this condition as closely approaching, if not actually, primary carcinoma of the liver. The surrounding area is markedly cirrhotic.

TREATMENT.—Medicinally nothing can be done for the cure of these new formations. When the growth or resultant cyst is solitary, removal has been accomplished successfully by surgery. For the multiple adenomata as a complication of cirrhosis or other disease, the treatment resolves itself into that for the causative malady.

Fibroma.—This form of tumor is composed of dense connective tissue. There may be no symptoms during life. The tumor may become large and give rise to biliary obstruction by pressure and death from cholangitis and cholemia. It may be possible in some cases to remove the tumor.

Angioma.—This variety of tumor has been also named nevus, cavernoma, and telangiectasis.

SYMPTOMATOLOGY.—They are usually multiple and vary in size up to that of a child's head. They sometimes consist of numerous connective tissue trabeculæ, enclosing communicating spaces containing blood. At other times there is a genuine increase in the arterioles.

DIAGNOSIS.—The diagnosis is difficult, as there are no pronounced signs or symptoms. A tumor causing a venous hum may justify a presumptive diagnosis. To remove all doubt a laparotomy may be necessary.

TREATMENT AND PROGNOSIS.—These angiomata have been successfully resected. When this can be done the prognosis is favorable. Generally, if the tumors are not large the outlook is good, even without operation.

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MALIGNANT TUMORS

Etiology, p. 207—Symptomatology, p. 207—Diagnosis, p. 209—Clinical varieties, p. 211—Treatment, p. 211—Prophylaxis, p. 211—Prognosis, p. 212—Pathology, p. 212.

Etiology.—The influence of age and sex has already been discussed. As at least 95 per cent. of all cases of malignant disease of the liver are secondary, the question of etiology comes to be one of the etiology of the primary growth in the breast, throat, stomach, rectum, kidney, and so on. In 85 per cent. of the cases of primary malignant diseases the growth is associated with cirrhosis, and will be dependent upon the same causes as produce that condition. In primary sarcoma of the liver, there may be some embryological abnormality in the arrangement of the tissue elements. An endodermal bud divides so as to form the gall-bladder and bile-ducts, the parenchyma of the liver, its blood-vessels, and the stroma of connective tissue. There may result from this an over-abundance of mesodermal material.

Symptomatology.—CLINICAL HISTORY.—*Mode of Onset.*—The onset may vary considerably. The disease may exist for some time in a latent condition, especially if it is primary and located in the central portion of the liver or in its upper and posterior aspects. Then, again, the clinical manifestations of the disease may be obscured by those of the primary growth. In massive primary malignant disease the growth is usually rapid and extensive, but less rapid in secondary examples.

Symptoms during Progress of Disease.—(*a*) *Objective Symptoms.*—Jaundice will be found to exist in slightly over 50 per cent. of the cases. Early in the disease there is emaciation, and the skin is dry, wrinkled, and of a dirty sallow color. The facial expression as the disease advances is one of a grave wasting illness with sunken eyes, hollow cheeks and temples, and haggard appearance. There is progressive absorption of the subcutaneous fat. The patient takes on the characteristic appearance called cachexia, which differs markedly from the appearance assumed in any other form of hepatic tumor. The liver is seen to move with respiration.

(*b*) *Subjective Symptoms.*—Loss of appetite and nausea soon come on, and in some cases there is vomiting. As the tumor increases in size a sense of fullness in the region of the liver becomes increasingly pronounced. Pain is present in varying degree extending to the right shoulder. There is a steady loss of strength.

PHYSICAL FINDINGS.—*Observation.*—As the disease advances, in most cases a bulging will be seen. In cases there may be considerable distention of the abdomen due to ascites. There is an apparent and progressive anemia, and, not infrequently, there is edema of the feet or general anasarca. The breathing may be embarrassed through involvement of the pleuræ or diaphragm, by extension to the thoracic glands, or the upward pressure of the enlarging liver. In advanced cases the nodules on the liver may be visible through the emaciated abdominal walls, and the superficial veins may be distended.

Palpation.—By the time the disease has made such advancement as to give rise to distinct clinical manifestations, some enlargement can usually be made out. If the tumor is in the left lobe it will be felt in the epigastrium, but if it is in the right lobe the mass will be in the right hypochondrium. The border of the liver will project below the rib margin for some distance. The border of the liver is generally felt to be uneven, and the surface of the liver to be studded with nodules, some of which present a depression. In case of diffuse cancerous infiltration the surface of the liver may be smooth and hard. There is commonly enlargement of the lymph-glands in the groin, the neck, and above the clavicle. The supraclavicular glands are sometimes markedly enlarged through metastasis in cases of cancer in the abdomen.

Percussion.—The area of liver dullness can be mapped out and the rate of growth thereby determined. The degree of ascites, if present, is also noted, as well as any further growths in the thorax and abdomen.

Auscultation.—A venous hum or bruit may be heard. It may be caused by pressure on the inferior vena cava, by interference with the portal circulation from cancer nodules or large glands, or by the vascular condition of the liver. The growths—especially the numerous and more superficial ones of secondary cancer—may cause an inflammation of the capsule resulting in a friction sound.

LABORATORY FINDINGS.—If a primary tumor has been previously removed, it may have yielded positive evidence of malignancy.

Ascitic Fluid.—When a specimen of the ascitic fluid is obtained it will be found to contain leukocytes, sufficient in number to suggest the idea of purulency, though there is no peritonitis or real pus present. In the blood there is a reduction in the number of red-corpuscles and still greater reduction in the hemoglobin. There may be an increase in the number of leukocytes, though this is not frequent, and, when it does occur, is liable to be intermittent.

The Urine.—The urine is likely to be reduced in amount and is highly colored. When there is jaundice, but not complete obstruction of the common duct, urobilin will be found in the urine. Bile coloring matter will also be found in cases with jaundice. Albumin and indican may occasionally be present. Creatinin, absent from normal urine, is found in large quantities. In cases of melanotic sarcoma of the liver melanin usually appears in the urine, and can be detected by the addition of a solution of ferric chlorid which produces a black color. When voided, the urine may be dark brown or almost black due to the bile coloring or melanin, or become so on exposure to the air. Melanuria is of much value in deciding that the case is one of secondary sarcoma of the liver.

The Feces.—In jaundiced cases the feces will be clay-colored.

SPECIAL EXAMINATIONS.—The employment of the *x*-rays, or the screening of the patient, may reveal the presence of irregular enlargement of the liver and aid materially in removing doubt.

Diagnosis.—CLINICAL HISTORY.—The salient points in arriving at a diagnosis of malignant disease of the liver are enlargement of the organ with evidence of tumor in its substance, progressive emaciation and loss of strength, more or less pain in the liver extending to the shoulder and back, the condition of cachexia so peculiar to malignant disease, the ashy, dirty, or muddy color of the skin, the knowledge of a primary focus or of metastasis to some other part of the body, and the discovery of umbilicated nodules on the liver. The diagnosis is further strengthened by the onset of jaundice and ascites, by the discovery of friction, by the appearance of melanuria, by the occurrence of occasional fever and a steadily increasing anemia.

DIFFERENTIAL DIAGNOSIS.—*Hypertrophic Biliary Cirrhosis.*—In this condition there is no evidence of a primary growth; the liver is not as hard as in malignant disease; there is always jaundice; there is an absence of pain and cachexia; and the liver is not nodular.

Portal Cirrhosis.—There is usually an alcoholic history, the course of the disease is chronic, there is a proneness to the development of ascites, mucous membrane hemorrhages are frequent, and the nodules on the surface of the liver are small. Cases of nodular cancer of the liver with cirrhosis and atrophic cirrhosis with nodules may be indistinguishable.

Amyloid Liver.—There is the history of chronic suppuration, as in tuberculosis or syphilis; the spleen is enlarged; there is less rapid wasting; and the urine contains albumin and casts, which become blue under the iodin test.

Echinococcus.—The multiple cyst form of echinococcus disease may closely resemble the nodular form of malignant growth, but in it the progress is slow and there is an absence of the cachexia of the latter. In the hydatid disease the spleen is enlarged. There is no history of malignant disease elsewhere. One large hydatid cyst resembles massive malignant growth, but it occurs earlier in life than the tumor.

Syphilis.—A gumma may resemble a malignant growth, but there may be a history of spirochete infection. When there is doubt, antisyphilitic treatment should be prescribed. If the growth increases in size there need no longer be any doubt. The Wassermann test should be made.

Abcess.—When a malignant tumor grows rapidly or becomes softened in its center, the resemblance to an abcess in the liver may be close. In addition, in certain cases of cancer there is considerable fever, which renders it still more difficult to arrive at a definite diagnosis. A laparotomy may be required to remove the doubt.

Gall-stones.—A gall-stone in the common duct may cause symptoms that simulate cancer or sarcoma. There may be fever, jaundice, and pain in both. In the case of gall-stone obstruction the jaundice tends to lessen, whereas in the malignant condition it increases. In the gall-stone condition the liver tends to undergo obstructive cirrhosis and reduction in size. In malignant disease there is steady and rapid increase in size.

Primary and Secondary Growths.—In a number of cases the primary growth may remain latent, and the disease in the liver appear to be primary when in reality it is secondary. The following features of primary and secondary malignant disease of the liver will assist in making a correct diagnosis:

In the primary form of the disease there is a single tumor; the growth is very rapid; jaundice is rare; ascites are also rare; the course is quick; emaciation is less; and there is no evidence of previous malignant disease.

In the secondary involvement of the liver the following characteristics should be noted: There are multiple tumors; their growth is slower; jaundice is frequent; ascites is common; the course is more moderate; emaciation is rapid and extreme; and a primary focus can be discovered in many cases.

Primary malignancy is more frequent among men, and secondary malignancy among women, primary to secondary being in the ratio of about 1 to 30.

Carcinoma and Sarcoma.—In the examination of many lists of cases it appears that cancer of the liver is about eight times more frequent than sarcoma.

With regard to *sex,* cancer is found in 70 per cent. of the cases among males and sarcoma in 30 per cent., while among females the ratio is 59 per cent. sarcoma and 41 per cent. cancer.

As to *age* primary carcinoma is very rare under forty years of age, but sarcoma may occur at any age, and has been reported as being congenital. In the case of secondary malignant tumors in the liver, the age of sufferers from cancer is greater than from sarcoma.

As to the *location of the primary disease,* it should be noted that in the case of cancer in the liver the primary focus was some disease in the stomach, colon, esophagus, pancreas, gall-bladder and uterus, whereas in sarcoma the primary focus was in the suprarenals, mediastinum, bone, lung, or was melanotic and general. These entirely different locations of the primary foci assist in deciding the nature of the secondary growth.

Clinical Varieties.—As about 95 per cent. of all cases of malignant disease of the liver are secondary to malignant disease elsewhere, the clinical features must vary greatly. In the vast majority of the cases it becomes a question of cancer or sarcoma of the stomach, uterus, intestinal canal, kidney, or bone, etc., with the disease of the liver added on as a complication. Usually the secondary form of the disease is slower than the primary; but when the primary is of slow growth the secondary may attain great size before death occurs. In those cases where the primary growth is latent the dominant features are those due to the disease in the liver. Primary malignant disease in the liver, both carcinoma and sarcoma, may be of the following types: massive, nodular or multiple growths; diffuse or infiltrating; the form developing in a cirrhotic liver; and the melanotic. The growths may become invaded by microörganisms, and the clinical manifestations of sepsis result.

Treatment.—PROPHYLAXIS.—When one remembers that about 95 per cent. of the cases of malignant disease in the liver are secondary to the disease in some other part of the body, the question of prevention becomes mainly one of properly treating lumps in the breast, lacerations of the uterus, ulceration of the stomach, suspicious erosions of the lip, and so on. The avoidance of habits that induce cirrhosis, and the proper treatment of cholelithiasis are also important preventive measures.

GENERAL MANAGEMENT.—*Diet*—The diet should be of such a character as to be nutritious and yet easily digested. These patients, as a rule, do not desire meat, and should be given milk, milk foods, light puddings, cereals, plain bread, jellies, tea or coffee. Many of these cases find benefit from being allowed some alcoholic stimulants. L. D. Bulkley excludes meats, milk, coffee and alcohol.

Treatment of Symptoms.—*Pain* is best relieved by the administration of **morphin and atropin in combination**, preferably under the skin, as by this method there is less derangement of digestion. Such drugs as **aspirin, chloral, conium and bromids are of little value. A plaster containing extract of opium and belladonna** has been recommended, but a **hypodermic injection of morphin** is cleaner and decidedly more effective. The **local application of heat** has been tried. **A properly fitting belt** may support the liver and lessen the pain. Unnecessary examinations should be avoided.

When there is *jaundice* the pruritus may be troublesome, and may require **alkaline baths** or the **sponging** of the skin with a **carbolic acid lotion** in the proportion of 1 in 50 of water, or the prescribing of small doses, grains 5-10 (0.325-0.65 gram), of **choral hydrate**, or the employment of **morphin hypodermically.**

Vomiting, which may be very troublesome, should be treated with

bismuth, carbonate of magnesium, creosote, dilute hydrocyanic acid, butyl chloral hydrate, cocain, effervescent waters, bicarbonate of sodium, ice, mustard over the stomach and a hypodermic injection of morphin.

Constipation is treated by salines and gentle laxatives, as **cascara, phenolphthalein, or castor oil** in capsules. **Glycerin suppositories** may be given a trial. Mention should be made of the use of **potash salts** as recommended by Dr. Bulkley. He prefers the **acetate of potash.** This is combined with cascara, extract of rumex and nux vomica. Should *ascites* become marked, **tapping** may be required.

RADIUM AND *X*-RAY THERAPY.—Little can be hoped for from these agents. They cannot remove or control the advance of the disease. In one case the writer found some benefit for the relief of pain by the employment of *x*-rays.

SURGICAL TREATMENT.—The removal of the tumor has been performed a number of times, and with moderate success in some instances. If the operation is performed while the growth is small, and early in the attack, the prospects are relatively improved. Operation is futile when the disease is advanced, diffuse, multinodular, complicated by hepatic cirrhosis, or when there is an active primary growth in some other part of the body.

Prognosis.—In a few cases recoveries have been recorded where malignant disease of the liver was thought to have existed. It is conceivable but highly improbable. When the tumor is small and is removed early, there is a possibility of arrest in the progress of the disease, or even of a cure, in primary cases in the liver, or in cases extending to the liver from the gall-bladder. But the diagnosis must be made by an exploratory opening. If the disease has advanced sufficiently to be diagnosed without a laparotomy, the case is quite hopeless. The duration of life is brief. In primary malignant disease a fatal termination may occur in a few weeks, but is seldom prolonged beyond six months. When the disease is secondary, the outlook depends a good deal upon the activity of the primary tumor and its location, but the average will be about six months. Should the primary tumor be inactive, the patient may live a year or more after the liver has become involved. The onset of ascites or edema of the legs points to a near approach of the end.

Pathology.—MACROSCOPICAL FINDINGS.—The macroscopical appearances vary greatly. There may be only one tumor in the liver, which, again, may vary greatly in size; or there may be many new growths. These new growths may be primary in the liver, or secondary to the disease in some other organ, or they may be secondary in the liver to a primary one in the liver itself. These tumors may be very small or almost fill the entire abdomen. At times they are quite firm, but in some instances are soft in the center. They vary in color also, are yellowish, grayish, or greenish from bile-staining. Sometimes the nodules are almost white. Some livers reveal the appearance of cirrhosis, their surfaces being studded with nodules—a condition of malignant hobnail. There is also, in some cases, a manifest degree of perihepatitis.

MICROSCOPICAL FINDINGS.—Microscopically *primary cancer of the liver* is of three types: those arising from the liver-cells, those from the bile-ducts within the liver, and those that are a combination of these two. In the liver-cell type there is a hyperplasia of cells, which form nodules, and crowd upon adjacent liver tissue. The irritation appears first to arrange certain cells into a hepatic cord, around which the hyperplastic cells are grouped. The type arising from the intrahepatic biliary ducts is specially met with in hepatic cirrhosis. There is a proliferation of the bile-ducts, resulting in malignant cholangioma. In cirrhotic livers this multiplication of biliary ducts may be accompanied by a hyperplasia of the liver-cells, and the mixed form is produced. The cells in the liver, as in other organs, accumulate into the characteristic rests. When the cancer is secondary in the liver, the new growth maintains the histological features of the primary one.

Sarcoma of the liver is of three types: angiosarcoma, alveolar sarcoma, and the spindle or round-cell sarcoma. Of these types the spindle or round-cell variety is the only one that appears to arise from mesoblastic tissue and be a true sarcoma. It is very doubtful whether there are any instances of pure angiosarcoma or alveolar sarcoma unmixed with the cellular type.

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INFECTIOUS JAUNDICE—WEIL'S DISEASE

Etiology.—This is a disease of young adults, affecting males and females in the ratio of 9:1. It is very rare among children, though cases have occurred. It is caused by the use of infected water and meat, but is not contagious. Infected animals, ducks, and geese may pollute water used for domestic and bathing purposes. The meat of diseased animals has communicated the infection. Epidemics have been traced to the water supply, and one was found to have been caused by using the water of a river frequented by infected ducks. Soldiers, butchers, and sewermen suffer most frequently. The disease prevails in the hot months of the year. While insanitary conditions are important as factors in the etiology of the disease, the real cause is an infective organism. Weil, Jaeger, Satterlee, Brooks, Rolleston, Libman and others are agreed that the *Bacillus proteus fluorescens* is the causative organism. Others contend that a spirochete is the cause, while J. M. Anders is of the opinion that different organisms may be the exciting agents. Inada and his co-workers regard the *Leptospira icterohæmorrhagica* as the cause.

Symptomatology.—CLINICAL HISTORY.—The onset of the disease is sudden and marked by fever, rigors, malaise, loss of appetite, weakness, diarrhea, headache, thirst, dizziness, pains in the back and limbs, disturbed sleep, delirium and drowsiness. After the third day there is jaundice. The urine is dark and contains bile. Sometimes there is vomiting, and constipation may take the place of diarrhea. The tongue is coated. There may be abdominal pains. Improvement usually occurs from the fifth to the tenth day, and after a few days of convalescence there may be a relapse with a return of the symptoms. The jaundice may last about two weeks and recovery is slow, there being marked debility. The pains in the back and limbs may be so severe as to overshadow the other symptoms. The muscles may be extremely tender to pressure, and the patient may find it very difficult to make any movement. There may be various skin disturbances, as herpes, urticaria, or erythema. Epistaxis, hemoptysis or other hemorrhages may occur.

PHYSICAL FINDINGS.—Early in the attack the liver becomes enlarged and is tender to pressure. The spleen is also enlarged. The abdomen may be tender to pressure. The temperature rises above normal with the invasion of the disease, and reaches 104° F. (40° C.). With the subsidence of the disease it gradually falls, reaching normal about the tenth or twelfth day. The pulse is weak and varies from 110 to 120 or more per minute; but as the jaundice deepens it generally becomes less frequent. The presence of nephritis, which is constant in the disease, is revealed by the urine containing albumin, casts, and often blood. It also contains bile and bile acids. The nephritis is concurrent with the enlargement of the spleen, and the albumin disappears from the urine slowly.

Laboratory Findings.—Bile coloring, bile acids, casts, and blood-corpuscles are found in the urine. The most important finding is the presence in the viscera and the urine of the *Bacillus proteus fluorescens*. When this organism is cultured and injected into animals, it gives rise to acute degenerative changes in the liver and kidneys. This organism is not found in other forms of jaundice.

Diagnosis.—An acute febrile disease with jaundice, severe muscular pains, enlarged liver and spleen, occurring in epidemics, should enable one to make a diagnosis. It can be distinguished from relapsing fever by the presence of the *Spirochæta obermeieri* in the latter. The surroundings should differentiate it from yellow fever. The reduced liver dullness of acute yellow atrophy contrasts with the large liver of Weil's disease. Typhoid fever rarely presents the degree of jaundice of this disease, and the finding of *Bacillus typhosus* should clear up any doubt. The sudden onset in Weil's disease and the gradual invasion of typhoid fever should also be remembered. Catarrhal jaundice is a local disease, while infective jaundice is a general disease with considerable fever, and a number of cases occur at the same time. No doubt some fatal cases of Weil's disease have been regarded as acute yellow atrophy, while some of the cases that have been diagnosed as the latter disease, ending in recovery, have been examples of Weil's disease.

Treatment.—The treatment is the same as that for catarrhal jaundice. **Rest in bed, a milk diet,** and the drinking of **water** freely. Large enemata have been recommended. A gentle **saline aperient** when there is constipation should be given. Mild intestinal antiseptics find favor with some, such as **calomel,** grain 1/20 (0.0033 gram), **salol,** grains 5 (0.325 gram), **beta-naphthol,** grains 3-10 (0.195-0.65 gram), **bismuth salicylate,** grains 5-15 (0.325-1 gram), or **thymol,** grain ½-2 (0.032-0.13 gram). **Alcoholic beverages** should be **forbidden.** Inada and his associates have recommended **immune serum.** It is given intravenously in doses of minims 7 (0.42 c.c.) per kilo body weight. It is effective only if given in the early stage of the disease. The subcutaneous method is not so satisfactory.

Prognosis.—The mortality varies with the locality, the race, the severity of the attack. It may vary in different epidemics; thus it may range from 10 to 60 per cent. In Europe it is less severe than in Asia and Egypt.

Pathology.—The liver is generally enlarged and is bile-stained; there is cloudy swelling of the liver cells; there may be degenerative changes and fatty metamorphosis, similar to that found in acute yellow atrophy, and the mucous membrane of the ducts may give evidence of degeneration. The spleen is enlarged and may contain hemorrhages. The kidneys are stained with bile-pigment; the cells are undergoing cloudy swelling, and there is tubal nephritis. Hemorrhages may be present in the skin in various organs, and in the mucous and serous membranes. The heart-muscle is weakened, and softening and cloudy swelling have been observed in fatal cases.

Historical Summary and Distribution.—In 1865 Weiss described an epidemic of jaundice which he called "infective jaundice." In 1886 Weil gave an account of some cases of jaundice under the heading "A Peculiar Form of Acute Infectious Disease Characterized by Jaundice, swelling of Spleen, and Nephritis." The paper attracted much attention; but the author was in doubt whether to regard the condition as a modification of other infections or as a new disease. Most French and a considerable number of English writers hold to the former view. The opinion is steadily gaining ground that it is caused by the *Bacillus proteus*. It is more prevalent around the Mediterranean Sea than elsewhere, and the Greeks are specially liable to it. It has also been called "febrile jaundice," "bilious typhoid," and "icterus epidemicus." It has been known to exist since the beginning of the eighteenth century.

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ACUTE YELLOW ATROPHY OF THE LIVER

(*Icterus Gravis, Typhoid Icterus, Acute Parenchymatous Hepatitis, Essential Hemorrhagic Icterus, Parenchymatous Degeneration of the Liver*)

Etiology.—PREDISPOSING CAUSES.—*Sex.*—In the case of children, males are attacked more frequently than females, the ratio being about 3:1. In the case of adults, females suffer twice as frequently as males.

Age.—Half the number of cases occur between the ages of 20 and 30 years of age, and four-fifths between 10 and 40 years of age. Slightly under one-fifth of the cases appear in persons less than ten years old. The subacute type is generally met with among children.

Worry.—A certain number of cases of acute yellow atrophy of the liver have been preceded by mental anxiety; but it has not been established that this occurs sufficiently often to make it bear the relationship of an actual predisposing cause.

Pregnancy.—It is admitted that pregnancy is a predisposing factor in the etiology of the disease. Pregnancy appears to render the liver very susceptible to morbid influences and changes. The disease usually appears after the seventh month of pregnancy.

Alcoholism.—The disease has been observed in some cases to follow both the acute and chronic abuse of alcoholic liquors. The abuse of alcohol may lower the vitality of the liver cells and render them more liable to be attacked by other toxic agencies, making the development of yellow atrophy possible.

Infections.—The disease has been observed to follow influenza, typhoid fever and syphilis. It is most likely that these infections reduce the resistance of the liver to toxins produced in the intestinal canal. It has not been proven that any of these infections do more than act as predisposing influences. The real infection causing the disease may in this way be given an opportunity to attack the liver.

Liver Disease.—There is a well-marked connection between this disease and previous and existing disease of the liver. Yellow atrophy may in this way be a terminal phase of catarrhal jaundice, cirrhoses, gall-stones, venous engorgement, or hepatitis. The impaired condition of the liver under such circumstances renders it liable to an attack by streptococcic infection.

Toxic Agents.—It is well known that chloroform, ethyl chlorid, ether, and phosphorus, may act injuriously upon the liver. Poisonous doses of these agents have been followed by jaundice, but there are reasons for regarding such a condition as differing from true yellow atrophy. At most, they could only be predisposing agents. The combined action of chloroform and certain bacteria has caused a condition very similar to yellow atrophy. The jaundice of phosphorus-poisoning very closely resembles yellow atrophy, and it has been held by many that the two conditions are identical. This is not correct, as there are differences in the morbid anatomy and the clinical course of these two types of jaundice.

EXCITING CAUSE.—Several views have been set forth as to the exciting causes of acute yellow atrophy. One of these is that it is an acute inflammation of the liver, causing destruction of the secreting elements. Another view is that it is a general disease in which the breaking down and necrosis of the hepatic cells is only an incident or result. Still another view is that it is a disease *sui generis,* and the result of a special infection that kills the liver cells, enabling their own ferments to dissolve them—a true autolysis. This view explains the clinical course of the disease, and the pathological findings. In all other forms of jaundice produced by toxic agents there is enlargement of the liver, whereas in acute yellow atrophy the special feature is rapid reduction in the size of the organ, due to the disappearance of its cells.

What this special infection is has not yet been determined. Various organisms have been found in the liver of those dying of acute yellow atrophy, but none of them can be regarded as causative. The colon bacillus has been most frequently found, but staphylococci, pneumococci, and streptococci have also been present. All of these, however, are found in cases of disease of the liver without the clinical and pathological findings of acute yellow atrophy. It would, therefore, seem that search must be made for some other bacterium, or coccus. Along this line must also be sought a true therapy.

Route of Infection.—This is not known, but the weight of evidence favors the intestinal canal as the primary seat of infection, and the portal circulation as the carrier. This is borne out by the absence of any apparent point of primary inoculation, such as the tonsils, or a wound anywhere. The mode of onset and the progress of the illness point to an acute infection of intestinal origin.

Symptomatology.—CLINICAL HISTORY.—*Mode of Onset.*—The mode of onset is much the same as in an ordinary attack of catarrhal jaundice. There is loss of appetite, a feeling of malaise, usually some vomiting and constipation, a slight rise in temperature, some tenderness over the liver on pressure, the tongue is coated, and in a day or two jaundice makes its appearance. The pulse is slow—60 to 70 per minute—and the respirations normal. This condition lasts for a variable period; but at the end of a week or less to several weeks there develop symptoms of a very serious character. The patient becomes drowsy or comatose, may be very noisy or delirious, even maniacal, and the pupils are widely dilated. The jaundice becomes deeper, and the color may be greenish, the tongue is dry and brown, the vomiting is severe and repeated, and the pulse becomes frequent and weak. The respirations become quickened, and, as the case progresses, the vomiting may become almost continuous, the temperature usually falling below normal, though it sometimes rises above 105° F. (40.5° C.). The stools are dark and offensive, and both the vomit and the feces frequently contain blood.

The patient passes into a state of muttering delirium which may terminate in coma or convulsions. It should be noted that some cases run a chronic course of several months.

Symptoms During Progress of Disease.—Objective Symptoms.—The appearance of the patient clearly indicates his diseased condition, and this condition can be readily recognized by the listless attitude or the delirious actions which are often apparent. The onset of jaundice is an early feature of the disease, and resembles jaundice from other causes at first, but, as the disease passes into the second stage, the color changes and becomes greenish. The urine is dark and contains bile coloring matter and gives a reaction with Gmelin's test; but as the disease becomes more serious and enters the terminal stage, the urine usually becomes very dark; the reaction to tests for bile is frequently faint, or may be absent. The coated tongue and vomiting, which is very constant, and may contain blood, will attract attention. Toward the end diarrhea may occur, the stools containing blood and bile. There may be muscular twitching, subsultus, spasm, or paralyses, and there may also be squint.

Subjective Symptoms.—From the inception of the attack there is marked discomfort in the epigastrium, nausea, and malaise. These are often very frequent in the early stage and constantly present in the later stage. There is a feeling of intense depression, which is lost in the increasing delirium and mental derangement. The patient does not complain of pain, except headache, which usually is intense in the late stage. Marked photophobia often develops. Muscular pains may be present.

PHYSICAL FINDINGS.—(*a*) *Observation.*—The very ill appearance of the patient together with the jaundice should direct the attention to the true condition during the first few days of the attack. By the end of a week the change of color to a greenish tint and the presence of pronounced nervous symptoms, such as delirium, muscular movements, stupor, coma, mania, or the typhoid state, remove all doubt regarding the nature of the disease. The development of symptoms closely resembling those of meningitis adds to the certainty of the diagnosis. The tongue becomes dry, brown, and tremulous, and the teeth are covered with sordes. Strabismus may occur and, as the attack goes on, the pupils become very widely dilated. The respirations gradually increase in frequency, become irregular, and, finally, stertorous. There may be a reddish rash on the skin, and subcutaneous hemorrhages are not infrequent. There may also be epistaxis and swelling of the joints. Other hemorrhages, such as those of the retina, hematuria, or metrorrhagia may be met with, and pregnant women abort.

(*b*) *Palpation and Percussion.*—The pulse in the early stage is usually slow—about 60 to 70—but later on it becomes weak, quick, and frequent. There is generally some degree of epigastric tenderness on pressure. As the disease progresses, and especially in the second stage, the area of liver dullness is reduced and may disappear entirely. This reduction in the area of dullness may occur very rapidly in the grave stage of the disease, inasmuch as it decreases from five or six inches in health to one or two inches. In rare instances the liver becomes enlarged before the stage of shrinkage begins. In cases of yellow atrophy supervening upon disease of the liver with enlargement, the disappearance of hepatic dullness does not occur to the same extent as in ordinary cases, though even in these there is some reduction. Liver tenderness may become very marked in the grave period of the disease. The lessening of the area of dullness is due to reduction in the size of the liver which begins in the left side, and to the softened condition of the organ, permitting it to recede from the body wall and allowing the intestines to intervene. Flatulence occurs in all cases and in most it becomes a prominent feature, rendering the mapping out of the liver difficult. The spleen is found enlarged in 50 per cent. of the cases.

LABORATORY FINDINGS.—*The Blood.*—In the blood an excess of the amino-acids is found as well as in the urine. Tyrosin has been found in larger amounts than the breaking down of the liver would warrant; and it has been thought that its presence is due to the failure of the liver to deal with the protein bodies brought to it. The concentration of the blood gives rise to an apparent increase in the number of red corpuscles; and sometimes there is a moderate leukocytosis. Bile-pigments have been found in the blood and its coagulation time delayed. The colon bacillus has sometimes been found in the blood; otherwise its bacteriology has been negative. The alkalinity of the blood is lowered.

The Urine.—The urine is generally reduced in amount and varies in specific gravity from 1.015 to 1.030. It is dark and gives a reaction for bile-pigments, and contains an excess of urates. The dark color of the urine may be caused by other than bile-pigments; *i.e.*, Gmelin's test may prove negative, the coloring agent being urobilin. Bile acids have been found in some cases, but they are usually present only in traces, and may be altogether absent. Sugar is not found, but albumin and casts are often present in small quantities. The nitrogen excretion in the urine may be normal, increased, or diminished. The fall may be due to a lessened intake of proteins, and the increase may be the result of disintegration of the liver cells. The urea is decreased and the ammonia is increased. The urea should eliminate in health 80-90 per cent. of the nitrogen of the urine, but in yellow atrophy the amount may fall to 60 or even a lower percentage. The ammonia rises from the normal 5 per cent. to 20-35 per cent. It is highly probable that the ammonia is carried out in combination with acids and is not used for the formation of urea. Leucin and tyrosin were at one time regarded as very significant of yellow atrophy; it is now recognized that both are present in the urine in about 50 per cent. of the cases, and that one or the other is present in about 72 per cent. They are both absent from the urine in about 28 per cent. of the cases. When absent from the urine, however, they are found in the liver. It may be necessary to concentrate the urine to obtain these bodies. They are formed by the breaking down of the liver cells. When uric acid is found in excess it is due to the hydrolysis of nuclear tissue while the dissolution of necrotic tissue is taking place. The amino-acids are found in excess in the urine.

The Vomit and Feces.—These very frequently contain blood. Bile may also be found in the vomit. In the feces bile is always present, as the biliary tubules are not so completely occluded as to prevent a certain amount of the bile reaching the intestinal canal.

Diagnosis.—The acute onset of jaundice without a history of obstruction, the rapid increase in the symptoms, and the severe nervous disturbances that develop about the end of the first week, accompanied by violent vomiting, are sufficient to justify the diagnosis of acute yellow atrophy.

Care should be taken to distinguish it from the *icterus gravis* as a terminal stage of other forms of jaundice, and the *jaundice caused by phosphorus-poisoning.* In these varieties of jaundice there is enlargement of the liver, whereas in acute yellow atrophy the area of liver dullness is lessened. In phosphorus-poisoning the gastric symptoms are severe at the beginning, but in yellow atrophy they come on later. In phosphorus-poisoning the initial period is followed by a more or less quiescent period before the jaundice sets in. In yellow atrophy there is no such period. Phosphorus may be found in the contents of the stomach.

Association with Other Diseases.—Acute yellow atrophy may be a terminal stage of other types of jaundice, such as that caused by syphilitic disease of the liver, the jaundice occurring during pregnancy, or in cases of unrelieved obstruction, or in chronic cases of catarrhal inflammation of the biliary tubes. The spleen may be enlarged and softened, the heart-muscle is softened and shows cloudy swelling, the gastro-intestinal tract is frequently inflamed, the lymph-glands in the fissure may be enlarged, the meninges often reveal hemorrhages, and the kidneys are swollen and bile-stained.

Treatment.—PROPHYLAXIS.—With a view of lessening the liability to acute yellow atrophy, all forms of jaundice should be carefully treated, with the object of shortening their duration and controlling toxemia. Each form should receive appropriate treatment. Cases associated with syphilis must be given specific treatment. Cases caused by obstruction, and chronic catarrhal cases ought to be given the benefit of surgical measures. Cases occurring during pregnancy may call for the serious consideration of terminating gestation. As a further means of obviating the onset of icterus gravis or acute yellow atrophy, all cases of jaundice should be confined to bed for a time and restricted to a suitable diet, and the alimentary canal regulated. The general care of acute yellow atrophy does not differ from that for jaundice arising in connection with other diseases.

General Management.—*Diet.*—Milk in some form must be the essential in the diet. It may be peptonized, or made into junket, or whey may be given, or buttermilk may be tried. It may be well diluted with some effervescent water, or flavored with tea or coffee. Gruels may also be tried and these may be predigested. Some of the prepared foods, such as are used for children and invalids, may also be ordered. Sugar and chocolate may help to prevent or lessen the acidosis. Mild alkaline waters should be used freely, such as Vichy, and ordinary plain water should be drunk in as large amounts as possible. When the stomach rejects all nourishment recourse should be had to enemata, and malted milk, whey, white of egg, and dextrose in turn may be employed. All acids should be avoided.

Treatment of Symptoms.—For the vomiting **dilute hydrocyanic acid, cocain** in small doses, or small amounts of **morphin** hypodermically may be tried. **Calomel,** grain 1/10 (0.006 gram) every hour for eight or ten doses, may relieve the nausea and vomiting. A large **sinapism** over the stomach and liver has been found useful. For the delirium and sleeplessness recourse should be had to **tepid sponging** and an **ice-bag** to the head. A small dose of **morphin** is preferable to the bromids, veronal or trional by mouth. **Chloral** and all drugs yielding chloroform *should be avoided.* **Veronal** may be given in doses of grains 5 (0.325 gram) repeated as required. The constipation is best remedied by moderate doses of **cascara, some saline, or calomel. Large bowel washes** will relieve the constipation and also help to lessen auto-intoxication.

Specific Therapy.—There is no specific treatment known. As a means of preventing autolysis of the liver cells **horse serum** has been tried and found helpful. With the object of reducing the risk from acidosis, **bicarbonate of soda** is recommended in dram doses (4 grams) three or four times daily by the mouth, or 3 drams (12 grams) in one pint of warm water by enema. This amount may also be given intravenously. A **saline solution** may be given subcutaneously or intramuscularly to control the toxemia.

Medicinal Treatment.—When they can be retained, **beta-naphthol,** grains 5 (0.325 gram), **urotropin,** grains 5-10 (0.325-0.65 gram), **salicylate of bismuth,** grains 5-15 (0.325-1 gram), **sal ammoniac,** grains 5 (0.325 gram), may be given as intestinal antiseptics. For the same purpose one-half to one pint of a sweetened mixture of **acetozone** (1 in 2,000 parts water) may be drunk daily.

Management of Convalescence.—When recovery does occur, the utmost care must be exercised to prevent a relapse or the passing into the prolonged subacute type. The environments should be the best, the diet selected with the greatest care, the functions of the body regulated, all excesses avoided, and a lengthy period of rest advised.

Prognosis.—The disease is not invariably fatal. This always leaves the possibility of a recovery with a much damaged liver, and the liability to relapses and death.

Pathology.—Macroscopical Findings.—The first thing that attracts notice is that the liver is greatly reduced in size. In many cases this reduction is almost to half the normal weight. When yellow atrophy is a terminal stage of some other form of jaundice in which there has been enlargement of the liver, this process of reduction is apparent. Fatal jaundice within this reduction in size is an icterus gravis termination, but is not true yellow atrophy. The left lobe usually suffers most. In consistency the liver is soft and flabby, and doubles readily upon itself. The surface is smooth, but wrinkled, and the capsule is loose, owing to the wasted condition of the liver substance. It can be easily peeled off. The surface is sometimes mottled with reddish areas. When kept for some time the surface is covered with leucin and tyrosin crystals. The surface, when removed from the body, has a greenish-yellow color. The cut surface is bright yellow and may present reddish-brown areas. The yellow is due to bile staining. The reddish-brown areas are firmer, and are the result of an effort at repair or regeneration, and the longer the patient lives the more marked this change becomes. In these areas the fat and dead cells have been absorbed, leaving the fibrous tissue and the blood-vessels. These areas are depressed below the level of the yellow areas. The gall-bladder contains bile often mixed with thick mucus, and the larger tubes frequently contain only thick mucus.

MICROSCOPICAL FINDINGS.—Scrapings from the cut surface reveal degenerated cells, and leucin, tyrosin, and xanthin crystals. First the liver cells become fatty and swollen. This is followed by bile staining, granular degeneration, and a breaking up of the nuclei. Capillary hemorrhages are seen. As the degeneration increases the cells cease to stain. The necrosis begins in the intermediate portion of the lobule and extends toward the intralobular vein. The small bile-ducts are inflamed and more or less occluded by an accumulation of proliferated epithelium, thick bile and mucus. At the margin of the lobules may be seen columns of cells arranged around tube-like structures. This is an attempt at regeneration. In the firmer red areas nothing can be found but the vessels and connective tissue enclosing a few nuclei and red blood-corpuscles. In some cases the necrosis is so complete that the section cannot be recognized as liver tissue. A small-celled infiltration may be noticed around the portal veins and extending into the margins of the lobules.

In those cases that run a chronic course, there is evidence of regeneration as well as of atrophy. There is a proliferation of liver cells and bile-ducts grouped into nodules that give the appearance seen in cirrhosis. The surface of the liver, in such cases, is studded with nodules of varying colors, yellow, or whitish, but are often greenish or brownish from bile staining. These nodules may become quite large, and in their formation are surrounded by fibrosis. No definite line can be drawn between these changes in subacute yellow atrophy and in acute cirrhosis. The morbid histology of the two diseases appears to be identical.

In cases that are not too acute the liver cells multiply by a process of division of their nuclei. These new cells are arranged in tubular columns, and assume the form of nodules through the hyperplasia of other tissues. There appears to be a hyperplasia both of the liver cells and of the interlobular bile-ducts, all of which is an attempt to make good what the disease has destroyed.

History and Distribution.—The disease was first described by Ballonius about 1600. Bright gave a good description of the condition in 1836, with an excellent illustration of the appearance of the liver. In 1842 Rokitansky named the disease "acute yellow atrophy." The disease is rare but met with in all countries.

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CHAPTER V

JAUNDICE

By Chas. A. Elliott, M.D.

Experimental work on the jaundice problem, p. 217—Postulations, p. 222—Classification of jaundice, p. 225—Discussion of types, p. 227—Diseases in which jaundice is a prominent symptom, p. 230—Catarrhal jaundice, p. 230—Icterus neonatorum, p. 230—Chronic hemolytic jaundice, p. 231—Pernicious anemia, p. 234—Hypertrophic biliary cirrhosis (Hanot's disease), p. 234—Yellow fever, p. 235—Acute infectious jaundice (Weil's disease), p. 236.

The term jaundice (icterus) has been used in clinical medicine to designate the abnormal yellow or greenish discoloration of the skin, mucous surfaces, tissues and fluids of the body by bile-pigment. This has usually been considered the result of obstruction of some sector of the bile-passages with reabsorption into the blood or lymph-stream of bile-pigment previously excreted by the liver-cells.

This conception may now be properly expanded to include those disturbances in the metabolism of the pigments of the hemoglobin-bile-pigment series (pigments containing the pyrrol nucleus) which result in a positive balance in retained pigment.

It is conceivable that metabolic disturbances of the pigments of this series may occur as a result of a deficiency in the elements forming them, resulting in a negative balance. The conception of a negative balance in bile-pigment is only theoretical, and no application of it has been made in clinical medicine. Such a negative balance, not being obvious or detectable by clinical tests so far devised, would elude the observation of the present day clinician.

Those disturbances which result in general pigmentation of the body, often associated with toxic symptoms (cholemia), due to gross obstruction of the bile-ducts (obstructive jaundice) are comparatively well known. But even here the rôle played by the various components of the reabsorbed whole bile (pigments, bile-salts, and other products of liver secretory activity) is not so well known. The disturbances resulting from more or less complete destruction of the functionating liver-cells, with jaundice due to more or less complete suppression of liver function, are little known from a physiological and chemical standpoint. The best-known examples of this type are those of the most severe grades, such as acute yellow atrophy and the jaundice of yellow fever, the general clinical and pathological manifestations of which have been known for years. Finally, there are disturbances in pigment metabolism resulting in general pigmentation of the body as the result of the liberation of great quantities of pigment of this series, yet without obstruction of the bile-passages and usually also without toxic symptoms (hemolytic icterus). These last disturbances are not so well known from a clinical standpoint, and the mechanism of their occurrence is still obscure.

Since 1847, when Virchow's first demonstration, i.e., that some of the pigment crystals in old hemorrhagic areas were identical with bilirubin, opened up the possibility of bile-pigment being of extrahepatic origin, experimental work with bile-pigment has been constantly in progress. At the present time the jaundice problem centers around the problem of pigment metabolism. Experimental work with bile-pigment has been exceedingly difficult, the results being often conflicting and contradictory and difficult of interpretation.

It is impossible to reconcile the old clinical conception that all jaundice is merely mechanical in nature, due to obstruction of some part of the bile-tract and reabsorption of bile-pigment in the liver, with the results of recent accurate clinical and experimental observations. For example, the theory of the pathogenesis of critical attacks of chronic hemolytic icterus was made to conform to the pathology of the autopsy room. Such attacks of jaundice, associated with a corresponding grade of acute anemia, have often been carefully described from a clinical standpoint. Tremendous amounts of bile-pigment have been found to be eliminated, largely by the liver as bile. The condition was formerly considered to be of hepatic origin, without account having been taken of the possibility of extrahepatic origin of the pigment or of the physiological and chemical processes which might have taken place in metabolism before the pigments reached the liver.

It is conceivable that a close relationship may exist between most, if not all, of the pigments of the body; a relationship may certainly be assumed between those of the hemoglobin-bile-pigment series. The trend of clinical and experimental observation would make such an hypothesis seem likely. That these chemical interchanges among the pigment bodies (pigment metabolism) are of fundamental importance to the economy, is evident from the importance of the function of one of the series, namely, hemoglobin. That their volume is large is evident from the amount of pigment thrown off in the stools, probably representing the daily turnover of pigment in the body.

The idea suggests itself that in dealing with bile-pigment we are dealing with only one of a chain of fundamental substances required in the metabolism of the body in general and not associated with the processes of digestion, as was formerly thought. This conception must include a consideration of the prepigment "building stones" and their synthesis into the pigments of the hemoglobin-bile-pigment series, as well as their catabolism and elimination from the body.

It is conceded that a complete and well-rounded clinical conception of jaundice in the broad sense, including a clinical knowledge of the metabolism of hemoglobin and bile-pigments with their deviations from the normal, is not possible at the present time; nor will it be possible until a better understanding of these processes has been established. On the other hand, a mass of experimental evidence has accumulated of which advantage should be taken in clinical medicine. Such evidence leads away from the conception of jaundice as limited to the obvious obstructive types, and toward a broader conception which includes the less-known types of jaundice associated with hemolytic processes, disturbances in splenicohepatic chemical relations, pigment metabolism and, finally, disturbances in liver function.

While the whole subject of jaundice is still in process of elucidation, an attempt may be made to formulate a working hypothesis for its clinical interpretation. It must be remembered that such an hypothesis based upon incomplete experimental data may be found inadequate when imperfectly understood, until fundamental questions of physiology are solved.

Experimental Work on the Jaundice Problem.—A presentation of the subject of jaundice at the present time, to be complete, must include the results of such experimental work as seems to throw light upon the problem. This applies particularly to the question of the metabolism of the bile-pigments and bile-acids, the extrahepatic origin of bile-pigment and the mechanism of the reabsorption of bile from the liver. While the amount of literature upon these subjects is enormous, much of it is reiteration and only a few facts are established. These should be brought to bear upon the clinical conception of jaundice. While much remains to be done before anything like a complete understanding of the physiology of the bile-pigments, and their deviations from the normal, is established, enough is known to render some of the accepted clinical ideas untenable.

Many *technical difficulties* are encountered in working on the jaundice problem. Bile-pigments are exceedingly difficult chemical substances with which to work. They are sensitive to light, heat, atmospheric conditions and, especially, to most reagents, such as oxidizing and reducing agents. They are difficult to obtain in the pure crystalline form. Most of them are not readily soluble and when dissolved are subject to rapid change.

The *chemical tests* for bile-pigments used in clinical medicine are unsatisfactory. They depend for the most part upon color phenomena produced by the oxidation of bilirubin, the degree and rapidity of which cannot be controlled. Such color tests cannot be applied in the quantitative estimation of bile-pigment, unless a large allowance is made for technical error. Simple matching of colors, as in the use of a colored wedge or colorimeter, is inaccurate, since the color of the specimen depends upon the degree of oxidation, which is allowed to proceed for an arbitrary period before comparison. As this oxidation is variable and readily affected by extrinsic factors, quantitative estimations depending upon it are probably not accurate.

The spectroscopic, quantitative tests for urobilin are likewise open to criticism. In preparing the specimen for examination, oxidation is allowed to continue for an arbitrary time, but other uncontrolled and even unknown factors affecting the degree of oxidation may vary in different specimens. The spectroscopic tests are open to the further criticism that the end-point, depending upon the extinction of the characteristic absorption band, is difficult to determine, and is at best only approximately correct.

Certain preconceived and apparently fixed ideas which have dominated the jaundice problem have delayed progress. The idea of the circulation of bile-pigment, for the existence of which there is not the slightest convincing evidence, has been generally accepted as a fact. The theory of the hepatogenous origin of all bile-pigment, whether or not correct, has directed attention to the liver, which does not lend itself readily to experimental research. And, finally, the idea of the intestinal origin of urobilin has been accepted as a fact, although the experimental results could hardly be interpreted as supporting such an hypothesis.

On account of the great diversity of the fields of research into which the jaundice problem enters, additional difficulties are encountered. These fields require a corresponding diversity of technical knowledge—for example, in regard to liver function, pigment and iron metabolism, blood destruction, the effect of osmotic disturbances of the red blood-cells, and the circulation of the bile. These and other problems, as yet incompletely understood, have a bearing upon the jaundice problem.

Until the function of the liver and the spleen, and the metabolism of the hemoglobin-bile-pigment series of pigments are better known, the clinical conception of jaundice must remain incomplete.

Virchow, in 1847, advanced the idea of the identity of hematoidin and bilirubin. He showed that the rhomboid tablets and needle-like crystals found in old hemorrhages gave a positive Gmelin test for bile-pigment. This fact has since been sufficiently substantiated, but a controversy arose and is still in progress, as to the importance of this finding in its relation to the jaundice problem. It demonstrates beyond any question the possibility of bile-pigment being formed from blood-pigment without the intervention of the liver. Virchow concluded that there were two types of jaundice, hepatogenous and hematogenous.

Vierordt, in 1873, first described and developed the spectroscopic method for the quantitative estimation of bile-pigment. In 1879 Vossius injected pure crystalline bilirubin intravenously into bile-fistula dogs whose common bile-ducts had been ligated and resected, and obtained an absolute and relative increase in both the amount of whole bile and bile-pigment. This finding was later substantiated by Tarchanoff, Stadelmann, Hoppe-Seyler, and, more recently, by Whipple and Hooper.

Tarchanoff, working under the direction of Hoppe-Seyler on bile-fistula dogs, found that bile-pigment excretion was much increased by the intravenous injection of distilled water and still more increased by the intravenous injection of bilirubin or hemoglobin solutions. In these experiments bile-pigment was not found in the urine. He concluded that the liver rapidly removes both bile-pigment and hemoglobin from the blood stream, excreting it as bile-pigment.

Stadelmann, in 1891, published an excellent monograph on icterus in which he summarized the jaundice problem up to that date. He repeated the experiments of Vossius and Tarchanoff, added much that was new as a result of his own experimental work along the line of the so-called hemolytic type of icterus (as in toluylendiamin poisoning, phosphorus poisoning, and in arseniureted-hydrogen poisoning), and finally formulated a theory as to the origin of jaundice. This monograph remains to-day one of the best presentations of the subject of jaundice that has appeared. While Stadelmann's experimental work has been substantiated by subsequent workers, his conclusions must be modified to some extent to conform to the results of later experiments. Stadelmann came to the following general conclusions:

(1) Hematoidin and bilirubin are identical. The pigment found in old hemorrhages is not all pure hematoidin.

(2) The secretion of bile-pigment and the secretion of bile-salts are two independent functions of the liver, with little or no relation to each other. When one is stimulated the other is apt to be depressed.

(3) The amount of whole bile secretion depends upon various factors aside from the functional activity of the liver-cell, such as blood-pressure, stimulation of the vasodilator nerves, increased circulation of blood, the administration of whole bile or bile-salts by mouth, and diet. The secretions of bile-pigment and bile-salts may or may not vary with the total amount of bile secreted and do not parallel each other. The administration of whole bile or bile-salts by mouth increases the secretion of bile, but the total amount of bile-pigment secreted remains indifferent. When abnormal amounts of bile-pigment are excreted, as in hemolytic icterus, phosphorus- and toluylendiamin-poisoning, the amount of bile excreted is apt to be greatly diminished, the bile then becoming a thick, tenacious secretion, "polycholie," which offers much resistance to its passage through the smaller bile-radicles, in which the pressure is known to be very low. This may, and probably does, result in the obstruction and reabsorption into the blood stream of bile-pigment as well as of other excretory matter. The production

of jaundice in certain types of hemolytic icterus is explained on this hypothesis, and it is concluded that such "hematogenous jaundice" is after all hepatogenous in origin.

(4) Both free hemoglobin and bile-pigment are rapidly excreted from the blood stream by the liver as bilirubin, the former requiring more time for its complete removal. Stadelmann expressed his belief, which he states he was not able to prove, that the change from hemoglobin to bilirubin occurs only in the liver. He was able to demonstrate the ability of the liver to excrete as bilirubin great quantities of hemoglobin from the blood-plasma, the amount of whole bile being meanwhile greatly diminished.

(5) In hemorrhagic states with hemoglobinemia and hemoglobinuria, free hemoglobin rarely if ever appears in the bile. From this fact it is concluded that the liberated hemoglobin is changed to bile-pigment by the liver-cells.

(6) The average bile-pigment excretion in dogs was estimated to be 0.01 gram (1/6 grain) per kilo per day.

(7) The color of the urine is not dependent upon the reabsorption of bile-pigment from the intestine. It is the same in bile-fistula dogs where the common bile-duct is obstructed, as it is in normal controls.

Minkowsky, in 1892, from studies of the pathology of toxic jaundice, came to the conclusion that jaundice results as an anomaly of liver function. Under these pathological conditions the bile is diverted by the liver-cells into the blood capillaries and not into the bile capillaries, reversing the normal order (parapedesis or paracholia).

Pick, in 1893, working on certain forms of hemolytic jaundice, concluded that the bile became so greatly thickened in these conditions that it of itself effectually obstructed the finer bile-passages.

Eppinger, in 1902, reported a study of the histologic changes in livers of various types of jaundice and came to the conclusion that all jaundice could be explained on a mechanical basis. He explained the sequence of events as follows:

(1) Stasis in the bile capillaries.

(2) Dilatation of the bile capillaries, especially at the ends, forming ampulla-like enlargements.

(3) Rupture of the dilated bile capillaries.

(4) Extravasation of bile into the perivascular lymph-spaces, first at the periphery of the lobule, later throughout the entire lobule.

(5) Disruption of the cell-cords by extravasated bile, isolating the liver-cells and separating them from their blood supply. Such isolated liver-cells finally become necrotic and appear in the specimens as débris.

(6) The extravasated bile finds its way from the perivascular lymph-spaces, through the lymph-channels by way of the thoracic duct into the blood stream.

In common bile-duct obstruction all bile capillaries are found to be dilated. In cirrhosis of the liver with jaundice only certain segments are dilated. In toxic jaundice, as in phosphorus-poisoning, inspissated bile itself effectually obstructs the finer bile capillaries, forming "bile thrombi" behind which the fine bile-radicles are dilated and rupture into the perivascular lymph-spaces.

Chauffard, in 1907, studied cases similar to those reported by Minkowski as hereditary splenomegaly with chronic icterus. He found that the globular resistance of the red-cells to hypotonic salt solution was reduced, that microcytes abounded in the blood, and that granules with typical staining properties were unduly prominent. Other French workers, notably Widal and his followers, then described cases of acquired hemolytic icterus, and the condition became speedily established.

Widal, in 1911, maintained that bilirubin might be of extrahepatic origin and stated that conception of dissociated jaundice in simple terms much as it is conceived to-day. The chief argument advanced against Stadelmann's theory explaining all jaundice on the basis of obstruction and reabsorption in the liver, is the fact that such reabsorption would have to be selective. This hypothesis is difficult to believe, for only bile-pigment and neither bile-salts nor cholesterin are found in the blood-serum in cases of hemolytic icterus. This fact, together with the demonstration of bile-pigment in old hemorrhages, the association of anemia with chronic hemolytic icterus, and the experimental work with hemolytic toxins, seems to indicate the existence of extrahepatic jaundice, and, therefore, of the extrahepatic production of bile-pigment.

Whipple, Hooper and their co-workers of the George Williams Hooper Foundation for Medical Research at the University of California are doing much experimental work on the jaundice problem, especially along the line of bile-pigment metabolism. The more important of their conclusions as published may be summarized as follows:

(1) Bile-pigment excretion can be influenced at will by modifying the diet. Carbohydrate feeding increases, and protein feeding decreases bile-pigment excretion. Bile-pigment formation is partly dependent upon the functional activity of the liver and, at least in part, independent of red blood-cell or hemoglobin disintegration.

(2) There is a close relationship between hemoglobin and bile-pigment. Hemoglobin may be rapidly transformed into bile-pigment in the subcutaneous tissues, serous sacs, and blood stream, independently of the agency of the liver.

(3) Hemoglobin and bile-pigment are rapidly eliminated from the blood stream by the liver, increasing the output of bile-pigment. Hemoglobin is not quantitatively eliminated as bile-pigment, as there are probably other factors involved besides the direct quantitative change of hemoglobin to bile-pigment. In anemic states, hemoglobin elimination as bile-pigment may be greatly diminished, but this is very inconstant.

(4) From experiments on Eck fistula dogs, under conditions simulating a diminished functional activity of the liver, probably due to lessened blood supply, it was found that these dogs reacted in the same way as normal dogs, except that the total amount of bile and bile-pigment was diminished. This suggests that the formation of bile-pigment may, at least in part, be dependent upon the functional activity of the liver.

(5) Bile-pigment excretion and bile-acid excretion are two independent functions of the liver which do not parallel each other. The amount of whole bile may be entirely independent of both.

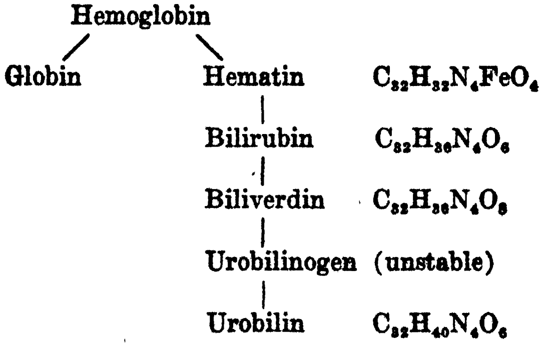
(6) The average bile-pigment excretion in experimental animals is 1 mg. (1/64 grain) per pound body weight, per six-hour period, with individual, hourly and daily variations.

(7) In obstructive jaundice, bile-pigment is reabsorbed from the liver by way of the blood capillaries. The lymphatic apparatus takes no essential part in this process, and is at best only a secondary factor in the mechanism of jaundice.

(8) Bile-acids and their salts are formed and excreted by the liver-cells. The amount of bile-acids excreted depends to a large extent upon the diet. On a standard diet a functionally deficient liver produces far less than the normal amount of bile.

Postulations.—It is impossible to correlate all of the conflicting ideas regarding the chemistry and physiology of bile-pigment and the mechanism of jaundice, but certain postulations may be made.

There is no question that there is a close chemical relationship between hemoglobin and the bile-pigments. This has been largely worked out by Hans Fischer, Frederick von Müller, Neubauer, and Wilbur and Addis. It may be roughly represented by the following diagram:


Hemoglobin
Globin
Hematin $C_{32}H_{32}N_4FeO_4$
Bilirubin $C_{32}H_{36}N_4O_6$
Biliverdin $C_{32}H_{36}N_4O_8$
Urobilinogen (unstable)
Urobilin $C_{32}H_{40}N_4O_6$

This diagram is offered as symbolical of the close relationship which exists between these substances, and not as indicating the actual change which takes place in the pigment turnover in the body. It is likely that the change from hemoglobin to bile-pigment is much more complex than is indicated by the diagram.

Hematin contains an iron molecule; the others do not. Bilirubin is crystallizable and may be obtained in the pure state. Urobilinogen and urobilin are names applied to groups of bodies, the former being very unstable. Hematin, hemosiderin, hematoidin, hematoporphyrin, bilirubin, biliverdin, bilifuscin, urobilinogen, urobilin, and stercobilin are all closely related; some are identical, and all are chemically and physiologically related to chlorophyl.

It is safe to say that the bile-pigments are all primarily blood-derived pigments, representing different stages in the cleavage of the hemoglobin molecule. Whether this change takes place in the tissues, circulation, spleen, or liver is not certainly known. Probably it takes place in all of these locations under varying conditions. Under normal conditions the liver excretes the pigment from the blood as bilirubin.

Urobilin, or its chromogen, urobilinogen, has been thought to be produced by the action of bacteria upon bilirubin in the intestinal tract, whence it is reabsorbed into the blood stream and later excreted by the kidneys. There are certain clinical observations that make it seem clear that urobilin may also be formed from hemoglobin in the blood stream without being reabsorbed from the intestinal tract. This seems to be the case during the acholuric crises of chronic hemolytic icterus, where great quantities of urobilin, and no bilirubin, appear to be liberated and excreted in the urine in a remarkably short period of time.

The pigments of the hemoglobin-bile-pigment series are of vital importance to the body, hemoglobin functionating as an oxygen carrier, and the bile-pigments being split products in its elimination. There is a more or less constant daily turnover of these pigments, the volume of which is large. It is roughly represented by the daily loss of pigment in the stools and urine. The synthesis of hemoglobin and its final excretion as bile-pigment is dependent, in part at least, upon the functional activity of the liver, and is much influenced by diet.

There is a constant disintegration in the blood stream of hemoglobin into less complex pigment bodies, or into other cleavage products not recognized as pigment. Such pigment is excreted, at least in part, by the liver as bilirubin, and by the kidneys as urobilin. Whether hemoglobin is quantitatively split up into bilirubin and urobilin, or whether bilirubin may be produced by processes other than simple cleavage of hemoglobin, is not known. Both of these possibilities seem reasonable.

A certain amount of bile-pigment is normally present in the blood-plasma (physiological jaundice). Some of the pigment which colors normal blood-serum yellow is known to be bilirubin, but whether or not it is entirely due to bile-pigment is still unsettled. Under abnormal circumstances yellow pigments other than bile-pigment are known to occur in the serum.

Both bile-pigment and hemoglobin are rapidly removed from the blood stream by the liver as bilirubin. If the amount of pigment liberated in the blood stream is greater than is readily taken care of by the excretory organs, it may accumulate in the plasma, and when the quantity is excessive, tissue jaundice results. Fixed tissues stained with bile-pigment may retain the pigment for a considerable length of time (vital staining).

In certain diseases associated with sudden and great transformation of hemoglobin into bile-pigment, the bile, as excreted by the liver, may become greatly thickened with pigment, and its viscidity alone may effectually block the passage of bile through the smaller bile-passages. The pressure in the finer bile capillaries under all conditions is very low.

The bile-acids (glycocholic and taurocholic acids) are excreted by the liver in the form of their sodium salts and serve in the process of digestion. The idea of the "circulation of the bile" as first promulgated by Schiff, in 1870, can no longer be accepted as far as it applies to the bile-pigments, as proven by Whipple and Hooper. Whether or not it may be accepted as applying to the bile-acids is still a question. It probably does not hold in either case.

The toxicity of the various substances contained in whole bile varies greatly. Bile-pigments are relatively non-toxic, bile-salts are more toxic, and whole bile is still more toxic. It is likely that the toxic manifestations collectively known as "cholemia" are due not only to the retention in the blood stream of bile-pigments, bile-salts and other excretory matter, but also, and probably largely, to liver insufficiency.

Obstruction of any part of the bile-tracts results in dilatation of the finer bile-radicles, stasis, and reabsorption of bile by way of the blood capillaries. Under such circumstances whole bile is reabsorbed, including such elements as bile-pigment, bile-salts, cholesterin, and other less-known substances, representing the total of liver excretory activity. Jaundice produced under these circumstances is "toxic," in the sense that certain symptoms are produced. These may vary greatly in degree, from such slight manifestations as skin itching with bradycardia and pigmentation, to grave comatose states and death.

In certain conditions, as in yellow fever and acute yellow atrophy, the liver-cells themselves are destroyed. The bile-pigment turnover is then arrested in the liver-cell, and bile-pigment is retained in the blood stream. Under these circumstances not only the excretion of pigment, but all of the many functions of the liver are impaired or completely interrupted, as, for instance, the excretion of the bile-salts, glycogen storage, and fibrinogen and urea formation. There is bile-pigment retention in the blood stream, although much is excreted by way of the kidneys. Bile-salts and other substances normally formed in the liver and excreted by it are excreted insufficiently or not at all, and a serious state of liver insufficiency results. Under these circumstances there may or may not be absorption from the liver, and it is of minor importance in comparison with the impairment of liver function.

Extremely rapid reparative processes, as a result of liver-cell regeneration, may occur following the destruction of liver-cells. Such regeneration, as estimated by Whipple and Hooper, may reach the extreme grade of from 100 to 150 grams (3 to 5 ounces) of liver-cells a day. Under these circumstances a functional equilibrium may be rapidly reëstablished and total or prolonged liver insufficiency avoided. This occurs in a great variety of pathological states.

Classification of Jaundice.—A final classification of jaundice cannot be made at the present time. Many points concerning the physiology of the liver and pigment chemistry, still under debate, must be settled before a permanent classification can be established.

If it is assumed that bilirubin originates in and is reabsorbed from the liver, as concluded by Stadelmann and Eppinger, the classification of jaundice seems simple enough. The problem is not so simple, however, since all recognize that the original source of such pigment, often liberated in great quantities, is not the liver, even though the final change to bile-pigment may occur there.

In a classification of jaundice the underlying causal factors for the liberation of bile-pigment should be taken into consideration. The question as to whether the final change from hemoglobin to bile-pigment occurs in the blood stream or in the liver is of less importance.

One type of jaundice is readily recognized as being due to obstruction of the bile-passages (obstructive jaundice). A second type occurs in which no mechanical obstruction of the bile-passages can be demonstrated. The liver, working at maximum efficiency, is inundated with pigment over and above what can be excreted (hematogenous jaundice). A third type may be recognized in which the liver is unable to perform its function, due to more or less complete injury, usually necrosis, of the liver-cells (suppression jaundice).

Classification

I. *Hepatogenous, Obstructive,* or *Choluric, Jaundice:*

Obstruction of bile-ducts; reabsorption of whole bile, containing bile-pigment, bile-salts, and excretory matter; bilirubinemia; bilirubinuria; cholemia.

1. Obstruction of the common bile-duct:
   - (*a*) Gall-stones, parasites, cicatrization, or congenital obliteration.
   - (*b*) Tumors of the common duct, Vater's papilla, or pancreas.
   - (*c*) Compression of the common duct by tumors of neighboring organs—stomach, kidney, colon, glands—or by aneurysm of the hepatic artery.
2. Obstruction of the bile-ducts and capillaries in the liver:
   - (*a*) Cirrhosis, malignancy, abscess, or syphilis of the liver.
   - (*b*) Toxic, infectious, or catarrhal changes in the ducts.
3. Passive congestion.

II. *Hematogenous* or *Acholuric Jaundice:*

Blood destruction with increased pigment liberation in the blood stream over and above what the liver excretes. No suppression of other liver functions; bile-salts and other excretory matter excreted as normal. Secreted bile thick with pigment; increase of bile-pigment in bile-ducts and intestinal tract. Non-toxic jaundice; acholuria; urobilinuria; anemia.

1. Blood alterations:
   - (*a*) Icterus neonatorum.
   - (*b*) Paroxysmal hemoglobinuria.
   - (*c*) Chronic hemolytic jaundice.
   - (*d*) Pernicious anemia.
   - (*e*) Leukemia.
2. Splenic alterations:
   - (*a*) Banti's disease.
   - (*b*) Hanot's biliary cirrhosis.
3. General infections:
   Septicemia, pneumonia, scarlet fever, typhoid, typhus, relapsing fever.
4. Parasitic infections:
   Malaria, ankylostomiasis.
5. Intoxications:
   Snake venom.

III. *Suppression* or *Toxic Jaundice:*

More or less complete injury of the liver-cells; suppression of liver function; pigment stasis in the liver; bile-salts not secreted or reabsorbed; other liver functions involved, such as urea and fibrinogen formation, glycogen storage, etc.

1. Acute yellow atrophy, eclampsia.
2. Phosphorus-, chloroform-, toluylendiamin-poisoning; trinitrotoluene-, tetrachlorethane-, and dinitrobenzol-poisoning in munition workers.
3. Yellow fever, epidemic jaundice.

A combination of two or more types of jaundice may be present in a single case. There are numerous possibilities. In obstructive jaundice inundation of the liver-cells with bile, separating them from their blood supply, may cause a widespread necrosis of the liver-cells. Under these circumstances a combination of obstructive and suppression jaundice may result. In hematogenous jaundice obstruction of the bile-passages by both viscid bile and pigment-stones is known to occur, producing a combination of hematogenous and obstructive jaundice. In suppression jaundice, with necrosis of the liver-cells and disruption of the cell-cords, more or less reabsorption from the liver probably occurs, giving a combination of suppression and obstructive jaundice. In all of these it is conceivable that blood dissolution may result in the cholemic state, adding the element of hematogenous jaundice to the type already present. Under any circumstances the identity of the predominant type of jaundice present in the specific case is usually evident.

Discussion of Types.—*Obstructive Jaundice.*—This is the best-known type of jaundice. Obstruction of the common bile-duct throughout its course or at the diverticulum of Vater, or even of the hepatic bile-ducts within the liver, may produce this type of icterus. This is usually due to such mechanical factors as stones, cicatricial contractures, malignancy, pressure upon the duct from without as by tumors of neighboring organs, or glands, or finally by catarrhal infections of the bile-ducts themselves. The latter, on account of the very low pressure within the bile-ducts, may effectually block the passage of all bile. As a result one finds dilatation of the small bile radicles, bile stasis, deposit of biliary thrombi in the bile capillaries, rupture of the bulbous ends and through the course of the bile capillaries, inundation of the liver parenchyma, and finally, reabsorption of whole bile by way of the blood capillaries. The inundation of the liver parenchyma produces grave consequences. The liver-cell cords are disrupted, liver-cells are isolated from their blood supply, much necrosis of liver-cells follows, and evidence of grave hepatic insufficiency may result.

In this type of jaundice all substances secreted by the liver are reabsorbed into the general circulation. Bilirubin and bile-salts appear in the blood serum and urine, and the tissues become intensely stained by bile-pigment. The exclusion of bile from the intestinal tract is not without deleterious effects, as has been shown by Whipple and Hooper. The stools become clay-colored. The urine contains large quantities of bilirubin and bile-salts, while urobilin is said to disappear. The urine is deep yellow or dark brown. Nephritis is apt to appear, evidenced by the presence of albumin and casts, suppression and uremia.

In cases of complete obstruction, symptoms of cholemia appear, such as: jaundice, bradycardia, skin itching, wasting, general malaise, somnolence, coma, and death. These symptoms are probably not solely due to the entrance of whole bile into the circulation; other factors are probably at work, of which acidosis, kidney and liver insufficiency, and the ill effects of excluding the bile from the intestinal tract, may be mentioned. A hemorrhagic state, said to result from the presence of bile-salts in the general circulation, or from the interference in fibrinogen formation by the liver, may develop. Widespread hemorrhages into the skin, subcutaneous tissues, muscles, or parenchymatous organs, hemoglobinemia and hemoglobinuria may result.

In incomplete obstruction, although only small amounts of bile seep into the intestinal tract, the symptoms may be much less severe. Under these circumstances most of the symptoms of cholemia usually fail to appear, even in the presence of obvious, and often intense, general pigmentation.

Hemolytic Jaundice.—All forms of jaundice due to extrahepatic influences are classed as hemolytic jaundice. The liver functionates normally, the bile-ducts are free from obstruction. All forms may occur, from mild cases with slight jaundice and no symptoms, up to the most severe grades of hemolysis, with intense jaundice. Hemoglobin is liberated and excreted as bilirubin by the liver, and as urobilin by the kidneys. It is assumed, but not proven, that this change occurs in the circulating blood, the liver and kidneys acting as excretory organs. Great quantities of bile-pigment are excreted by the liver, the bile becoming greatly thickened (pleiochromia). Stadelmann was of the opinion that the viscid bile effectually obstructed the biliary capillaries, and that reabsorption from the liver took place. The absence of bile-salts in the blood-plasma and urine speaks against this theory. The duodenal content includes a great deal of bile-pigment, is chocolate-colored, thick, and viscid. The amount of urobilin in the urine is greatly increased.

In certain types the fragility of the red blood-cells is greatly increased. This was thought by Troisier and Guillain to be due to the fixation of hemolysins to the red blood-cells, thus modifying the osmotic properties of their walls. Eppinger considered the increased hemolysis in certain types to be due to "hypersplenism." Splenectomy is known to cure chronic hemolytic icterus of the family type. In certain types of hemolytic icterus the spleen must, therefore, play an essential part in the process. The exact chemistry of this process is unknown.

Certain infections probably produce jaundice by causing hemolysis; examples of these are scarlet fever, pneumonia and streptococcic septicemia. In all of these the liver is also known to be affected. One or both factors may play a part in the production of such jaundice.

The symptoms of cholemia do not occur in hemolytic jaundice. There is no skin itching, bradycardia or bilirubinuria. Long-continued jaundice of this type is associated with severe anemia. In acute attacks (acholuric crises) associated with rapid dissolution of blood, such symptoms as restlessness, malaise, headache, vertigo, epigastric pain and tenderness, and acute swelling of the spleen, accompany the deepening jaundice. These symptoms, apparently produced by acute blood dissolution, resemble those of acute hemorrhage more than those of cholemia.

Suppression Jaundice.—A form of jaundice occurs in which there is more or less complete destruction of the liver-cells, with acute functional liver insufficiency. This is best known in such severe forms as yellow fever, phosphorus-poisoning, and acute yellow atrophy. Less severe forms due to scattered focal necrosis probably occur, but on account of the rapid regeneration of liver-cells functional disturbances do not appear except in the presence of extensive or complete liver injury.

In extensive liver injury, on account of the extremely acute and overwhelming onslaught, hepatic functional insufficiency develops. But in cases of severe liver injury, as in phosphorus-poisoning and yellow fever, regeneration may maintain liver function, and complete restoration results in the case of the patients who survive. In less extensive and acute injuries, hepatic regeneration maintains functional efficiency.

In extensive liver injury malaise, epigastric pain and tenderness, nausea, and vomiting, develop rapidly, and intense jaundice, hemorrhages from the gastro-intestinal tract, nephritis, bradycardia, stupor, coma and death may follow. The blood serum is intensely yellow, and contains large quantities of bilirubin with traces of bile-salts. The urine contains bilirubin in great quantities, bile-salts in traces, amino-acids, such as leucin and tyrosin, and albumin and casts in increasing quantities. Suppression of urine develops as death approaches.

A distinction between the symptoms due to cholemia and those due to acute hepatic insufficiency in these cases is impossible, except from a theoretical standpoint. The two conditions are closely associated but are not identical. Acute hepatic insufficiency causes interference in diversified chemical processes, such as urea and fibrinogen formation, glycogen storage, the secretion of bile for digestive action, etc.

Cholemia may be held in check by the vicarious excretion of waste products by way of the kidneys. Acute hepatic insufficiency may be held in check by the regeneration of the liver-cells. In either case the compensatory process may or may not be efficient.

At autopsy the liver is necrotic, smaller than normal, often nothing more than a ''mushy mass,'' as in acute yellow atrophy, or uniformly yellow and ''box-wood-like'' as in yellow fever. Extensive areas of regeneration may be present, as in phosphorus-poisoning. All of the above appear to be various phases of liver degeneration, producing a corresponding liver insufficiency.

The excretion of bile is augmented at first, but soon all secretion of bile through the biliary tracts is interrupted, as demonstrated by Stadelmann in phosphorus-poisoning, in which only a thin colorless mucoid secretion passes through the bile-duct.

Dissociated Jaundice.—The idea of dissociated jaundice has been a recent development of the French clinicians. In this country, Hoover and Blankenhorn have called attention to the subject. The conception of dissociated jaundice hinges upon the presence of either bile-pigment or bile-salts, separately or together, in the blood-plasma or urine.

Bile-pigment originates from hemoglobin in the circulation, and is excreted from the blood-plasma by the liver. Bile-salts originate only in the liver. In hemolytic icterus, bile-pigment alone appears in the plasma and urine. In obstructive jaundice (complete icterus) both bile-pigment and bile-salts are found in the plasma and urine. Bile-salts, being diffusable, are more readily excreted through the renal filter, and, following complete icterus, bile-pigment alone may be retained in the plasma (renal dissociation jaundice). The blood-plasma may retain bile-pigment without yielding it to either the urine or tissues, so that pigmental cholemia without bilirubinuria or tissue jaundice may result.

The term hepatic dissociated jaundice implies the diverting of either bile-pigment or bile-salts into the blood-plasma by the hepatic epithelium, without obstruction of the bile-passages. Whether or not this occurs is debatable.

In general these terms refer to various phases of the physiology of the liver and the hemoglobin-bilirubin series of pigments. Whether the finer distinctions implied by the term hepatic dissociated jaundice will come into general use depends upon the further development of our understanding of the physiology of the liver. Roughly speaking, the term dissociated jaundice, as generally used at present, refers to hematogenous jaundice.

Diseases in which Jaundice is a Prominent Symptom.—A few of the diseases usually considered as clinical entities are discussed from the point of view of interpretation of their jaundice manifestations:

CATARRHAL JAUNDICE.—This is a common type of jaundice, occurring sporadically, especially in young people. The jaundice is of the obstructive type. It is thought to be due to a catarrhal inflammation of the mucosa of the bile-ducts. On account of the very low pressure within the bile-passages, a relatively slight swelling of the mucosa of the ducts would effectually obstruct the passage of bile. It is thought by some to be a specific infectious disease, but a specific organism has not as yet been generally accepted as the causal agent. The possibility of catarrhal jaundice following a descending infection, by way of the blood stream, should be considered.

The symptoms are those of a mild gastro-intestinal disturbance with slight fever, followed by jaundice. Bile-pigment and bile-salts are found in large amounts in the blood-serum and urine. The stools are clay-colored and offensive. Constipation is usually present. The liver may be swollen and tender. The gall-bladder may be distended and palpable. Mild cholemic symptoms, such as malaise, stupor, skin itching and bradycardia, occur. These continue for several weeks. If the symptoms do not subside within a few weeks, or if they become severe, a mistake in diagnosis should be suspected, and other causes of obstruction should be sought. It is possible that a grave cholemic state, due to extensive liver-cell degeneration, may supervene in what in the beginning seemed to be a simple catarrhal jaundice.

ICTERUS NEONATORUM.—There are numerous conditions in which jaundice may occur in the new-born. Of these the following may be mentioned: congenital occlusion of the common bile-duct, septic infections, syphilis or cirrhosis of the liver, portal thrombosis, and catarrhal inflammation of the duodenum and bile-ducts. These types differ in no respect from those found in adults. The type commonly indicated by the term icterus neonatorum occurs in from 40 per cent. to 50 per cent. of the new-born. It is a mild type of jaundice, beginning about the third day following birth, and passing off in a week or less. It is apparently without symptoms or other manifestations. If the jaundice should continue, or become intense, the presence of one of the more serious conditions mentioned above should be suspected.

Many theories have been advanced as to the exact cause of icterus neonatorum. The following may be mentioned: (1) Increased destruction of red blood-cells immediately following birth; (2) obstruction of the common duct by mucus; (3) disturbances in the pressure within the bile-ducts, portal vein and hepatic artery, or stasis in the large abdominal veins, due to the change from the fetal to the post-natal type of circulation; (4) reabsorption of bile from the meconium.

Chronic Hemolytic Jaundice (*Chronic, family, or constitutional jaundice; acholuric jaundice*).—This is a remarkable disease, first described by Levy in 1898, by Hayem in 1898, by Minkowski in 1900 as hereditary splenomegaly with chronic icterus, and later carefully studied by Chauffard, and Widal and his co-workers in 1907. It is characterized by persistent jaundice, enlarged spleen, intense urobilinuria, increased fragility of the red blood-cells, anemia, and critical attacks with malaise and fever during which exacerbations of the jaundice and anemia occur.

It may be congenital, acquired or familial. In the *congenital type* a single member of a family may be affected from birth.

The *acquired type* usually appears in young adults. It is more severe; the crises are sometimes alarming, and it may occur as a manifestation of another disease process, such as chronic endocarditis, syphilis, or pregnancy, during the course of which pigmental disturbances associated with the phenomena of hemolytic icterus may arise.

In the *familial type* of the disease several members of the same or succeeding generations may be affected. (See Fig. 1.) It is usually the least severe type of the disease.

Chronic hemolytic jaundice is not rare. Most cases are mild, the jaundice being considered as an individual or constitutional peculiarity rather than as a disease. As Türk remarks, it may be considered little more than a "cosmetic blemish." When present it usually persists throughout life, not ordinarily interfering with the development or well-being of the individual. Only the unusual fulminating or severe cases attract serious attention.

Jaundice is the most obvious symptom and, except in critical attacks, is usually not intense. The discoloration is of light lemon color and varies considerably from time to time, but continues indefinitely. It is not associated with any of the toxic symptoms usually ascribed to cholemia. Much bile-pigment is excreted by the liver and kidneys. The duodenal contents are deeply stained with bile-pigment, and during the critical attacks, may be thick, viscid, chocolate-colored or almost black. The stools contain much bile-pigment but no blood. The urine is highly-colored, contains much urobilin and urobilinogen but no bilirubin or bile-salts.

The fragility of the red blood-cells is increased. This may be

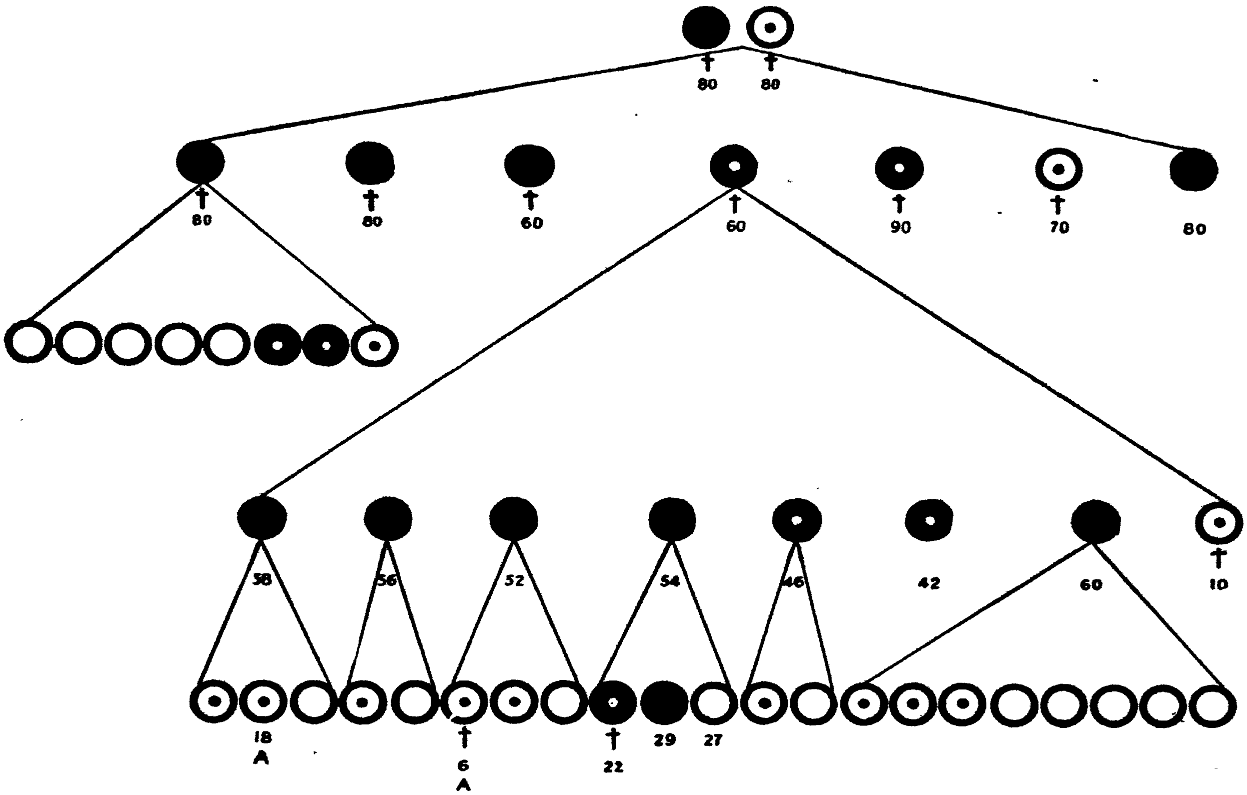


FIG. 1. FAMILY TYPE OF HEMOLYTIC ICTERUS.

The genealogical tree of the "B" family, in which the jaundiced members are represented by solid black figures. The cross indicates that the patient has died; circles with dots represent females; the figures denote age; "A" indicates intense anemia.

tested by adding washed red blood-cells to varying concentrations of hypotonic sodium chlorid solution. Hemolysis normally begins about 0.44 per cent. and is complete at 0.36 per cent. In chronic hemolytic icterus, hemolysis begins and is complete at higher concentrations, i.e., it begins at 0.6 per cent. or even 0.7 per cent. and is complete at 0.52 per cent. or 0.62 per cent. Hemolysis is reported to have occurred even as high as 0.82 per cent., and may occur in normal serum. In obstructive jaundice the fragility of the red-cells is decreased.

A moderate anemia of the secondary type is usually present, but this may become intense in severe cases, ultimately approaching the pernicious type. This appears to be due to the final exhaustion of the hematopoietic system. The white blood-cells are little affected. The blood serum contains much bile-pigment, as shown by an intense Gmelin reaction, but contains no bile-salts. Urobilin is constantly present in the urine, often in great quantities.

"Acholuric crises" occur occasionally throughout the course of the disease. These may vary greatly in severity, from little more than "bilious attacks" to intense and alarming periods of hemolysis, jaundice and anemia. Attacks usually last several days. They are characterized by malaise, depression, increasing jaundice, and deeply-pigmented stools and urine. Severe attacks may be fulminating, with rapid development of anemia and jaundice associated with fever, headache, vertigo, nausea, vomiting, and enlargement and tenderness of the spleen and liver. Hemoglobinemia may be present at the height of the crisis.

A large proportion of the red blood-cells may be destroyed during a severe crisis. As much as one-half of the total number of red-cells has been estimated to have been destroyed during a single crisis. Syncope or death may occur. The symptoms are not unlike those of acute hemorrhage.

The spleen is usually, although not always, enlarged. It may approach the size of a leukemic spleen. It increases noticeably in size during a hemolytic crisis, is tender to pressure, and may be quite painful. A perisplenic friction rub, or a systolic bruit may be present over certain areas of the tumor.

The liver may appear normal or slightly enlarged, and is sometimes tender during critical attacks. The bile is thick, and pigment-stones may be deposited in the gall-bladder. These may cause obstruction of the bile-ducts producing symptoms of obstructive jaundice associated with or independent of the symptoms of hemolytic icterus.

The *etiology* of this disease is still unknown. Chauffard and Widal consider it a disease of the red blood-cells associated with increased fragility of these cells. Eppinger looks upon it as "hypersplenism" in the sense of exaggeration of the hemolytic function of the spleen. It has been demonstrated that the spleen has an important, and probably essential rôle to play in the etiology of this disease. Banti first performed splenectomy for hemolytic icterus in 1903, reporting the operation in 1912. In 1915 Elliott and Kanavel were able to collect 48 cases of splenectomy for hemolytic icterus in the literature, including their own, the first reported case of operation in this country. Since that time many reports have confirmed the curative results of splenectomy in this disease. Because of the magnitude of the operation only severe manifestations should be considered as indications for splenectomy. Most of the cases are light or trivial.

PERNICIOUS ANEMIA.—The blood serum in many cases of pernicious anemia is stained deep yellow. This frequently gives a yellowish tint to the skin and mucous membranes of such patients. Some of these sera give a positive Gmelin test for bilirubin, and a few a positive Pettenkofer test for bile-salts on the dialysates of the sera. Whether the pigment is tenaciously held by the blood-plasma, not being given up to the tissues or excreted by the kidneys or liver, is not known. It may be pigment in transit, representing increased transformation of hemoglobin to bilirubin in the blood stream. A great increase is found in the urobilin in the urine and stools (stercobilin), and in bilirubin in the duodenal content. On this basis an increase in hemolysis is assumed to exist in pernicious anemia. The increase in the bile-pigments in the urine and stools sharply differentiates pernicious anemia from severe secondary anemias, in which the amount of bile-pigment excreted is greatly diminished.

The subject has attracted the attention of Addis, Hoover and Blankenhorn, Eppinger and Charnas, Schneider, Griffin, Sanford and Szlapka, and many others. The question as to the exact interpretation of the increased bile-pigment elimination found in these cases is still unsettled. Whipple is of the opinion that the hemoglobin may not be eliminated quantitatively as bile-pigment and, therefore, red blood-cell destruction may not be estimated on the basis of the amount of bile-pigment excreted. There are undoubtedly other factors which should be taken into consideration before the significance of the pigment changes occurring in pernicious anemia are well understood.

HYPERTROPHIC BILIARY CIRRHOSIS (*Hanot's Disease*).—This is a rare disease of long duration, usually beginning in childhood and continuing throughout life. It is characterized by enlargement of the liver and spleen, recurring febrile attacks with jaundice, but without signs of portal obstruction or ascites. The febrile attacks are associated with an exacerbation of all of the symptoms. The jaundice, when once established, continues throughout life. It may be slight in the intervals between the febrile attacks, but is intense at these periods. Bile-pigments and bile-salts are found in the blood serum, urine and stools.

In many respects it is a disease that closely simulates chronic hemolytic icterus; but the distinctive acholuric crises of the latter are wanting, although the febrile attacks with deepening jaundice superficially resemble them. Fragility of red blood-cells is not increased.

Splenectomy is said to have a beneficial effect on the course of the disease, but has not been performed frequently enough to establish its real value as a curative agent.

ACUTE YELLOW ATROPHY.—The term acute yellow atrophy is loosely applied to a group of cases in which there is an acute and widespread destruction of the liver parenchyma. Signs of acute hepatic insufficiency appear. Jaundice of the suppression type rapidly develops. The liver-cells are not able to excrete the pigment, which then accumulates back of the liver and appears in large amounts in the blood stream and urine. The fixed tissue-cells become pigmented. The bile-tracts are free. The manifestations of acute cholemia and acute hepatic insufficiency rapidly develop and a fatal termination results.

Just what the nature of this rare disease may be is unknown. There are features of it which suggest tissue sensitization with massive autolysis of the liver-cells.

YELLOW FEVER.—The author, serving as clinician of the Rockefeller Yellow Fever Commission * sent to Guayaquil, Ecuador, during the summer of 1918, had an opportunity of studying about 70 cases of yellow fever. The work was done under the auspices of the International Health Board. While working on this commission Noguchi found, in the blood and tissues of these cases, an organism which he was able to grow in pure culture. By inoculation of blood and tissue emulsions—as well as pure cultures obtained from human cases—into guinea pigs, he was able to produce a clinical picture and pathological lesions similar to those observed in the human cases. The organism was again recovered from the tissues of the experimental animals. The organism was provisionally called the *Leptospira icteroides.* Morphologically it resembles the *Spirochæta icterohæmorrhagiæ* of infectious jaundice, but is immunologically distinct.

The jaundice of yellow fever appears to be typically that of the suppression type. It is apparently similar to that of infectious jaundice, although more severe. It is a constant although variable manifestation of all cases. As a rule it varies with the severity of the disease, although it may be of an intensity in light cases out of proportion to the other manifestations of the disease. As a rule it appears on the second or third day, reaching its maximum about the tenth day and continuing well into convalescence. It may be intense after the patient has apparently recovered and seems otherwise quite well. The pigmentation first appears on the conjunctivæ at a time when the conjunctival surfaces are greatly congested, and later becomes more marked in contrast as the congestion disappears. It later appears in the skin and mucous surfaces.

The blood-plasma and urine are often markedly pigmented, out of proportion to the degree of pigmentation seen elsewhere, and they remain pigmented longer. The jaundice itself is non-toxic. Skin itching does not occur. At the end of convalescence, intensely jaundiced patients appear to be free from obvious toxic symptoms.

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*This commission was composed of Dr. Arthur I. Kendall, chairman; Dr. Hideyo Noguchi, bacteriologist; Dr. Mario G. Lebrado, Havana, sanitarian; Herman E. Radenbaugh, chemist; and Dr. Charles A. Elliott, clinician.

The jaundice in yellow fever is apparently of the dissociated type. The blood-serum gives a positive, often intense, Gmelin test for bilirubin and a negative, or at most, a weakly positive Pettenkofer test for bile-salts, on aqueous dialysates.

In the cases examined, the urine, which is always intensely pigmented, uniformly gave an intense Gmelin test for bilirubin and a negative, or at most, a faintly positive test for bile-salts. The foam of the urine was always definitely yellow. Some specimens of urine gave evidence of containing large amounts of urobilin.

In a few cases the stools appeared distinctly clay-colored, but most were dark brown, containing much blood.

The liver at autopsy is found to be profoundly affected in all cases. It is about normal in size and shape, although in some cases it appears to be smaller than normal. The larger bile-ducts appear free from obstruction. The surface of the liver is smooth, and ocher or deep brown in color. Some specimens are firm and cut with resistance, but others are friable and offer little resistance to the thumb when it is passed through the substance. The cut surface is dry, smooth, and uniform in color, the color varying from ocher to dark brown.

Microscopically the liver shows extreme fatty degeneration and necrosis of the cells and hemorrhage into the necrotic areas. In places all of the epithelial cells of the lobule appear to be necrotic; in other places there remain greatly swollen liver-cells filled with droplets of fat. The nucleus remaining at or near the center of the cell retains its staining properties fairly well. The necrosis and hemorrhage is most marked in the mid-zone of the lobule (hepatic artery zone), while both about the central vein and the periphery of the lobule the cells are less degenerated and may retain their staining properties fairly well. In some sections it is hard to distinguish the central vein on account of the extreme swelling and squeezing together of the surrounding liver-cells. In many cells the fat droplets are very numerous, some fine, others large and irregular, the nuclei retaining their position at or near the center of the cell.

In certain specimens there appears to be some interference in the absorption of bile-pigment, which is seen plugging the bile capillaries. In some specimens the Kupfer cells are seen to be packed with pigment. Glisson's capsule and the structures contained therein show much less change than do the liver-cells themselves.

From the clinical and postmortem evidence one must conclude that the degenerated liver of yellow fever is incapable of performing its function, that it not only acts as a barrier to the normal transformation of pigment in the body, but is also incapable of performing other functions such as, for instance, the secretion of bile-salts. The jaundice of yellow fever may therefore be considered primarily as a suppression jaundice, although there is undoubtedly some reabsorption due to bile capillary obstruction.

ACUTE INFECTIOUS JAUNDICE (*Weil's Disease*).—This is a specific infectious disease caused by the *Spirochæta icterohæmorrhagiæ.* The disease was first described by Weil in 1866. The *Spirochæta icterohæmorrhagiæ* was discovered and described as the specific organism causing the disease, by Inada, Ido and their co-workers in 1915. The disease occurs sporadically or in transient epidemics, usually in the lowlands, such as in the rice fields of Japan or in Flanders, where men lived under unsanitary conditions and in close proximity to rats. Wild rats are known to harbor the organism. It has been found in the kidneys and urine of from 10 per cent. to 40 per cent. of wild rats from widely separated countries. The rats seem to harbor the organism without serious trouble to themselves. The organism may invade the human body through the unabraded skin or the alimentary tract.

The onset of the symptoms is sudden, with fever, prostration, congestion of the skin and mucous membranes, headache, nausea, vomiting, epigastric pain and tenderness, jaundice, hemorrhages into the parenchymatous organs or from the mucous surfaces, albuminuria, nephritis, and uremia. The fever is high for one week and then subsides, although there may be a secondary rise.

The organisms invade the blood stream and parenchymatous organs early, but rapidly disappear. Few are to be found after the period of invasion. Coincident with the disappearance of the organism, antibodies appear and increase in the blood-serum.

The mortality of the disease varies in different localities, the figures showing 32 per cent. in Japan, 6 per cent. in Flanders.

Clinically the disease is similar to yellow fever, although there is less jaundice and more hemorrhage in infectious jaundice. Epidemically the two diseases differ in that yellow fever is transmitted by the mosquito *Aëdes calopus* Meigs (*Stegomyia calopus*) while epidemic jaundice is probably transmitted directly through the skin or alimentary tract from the urine of wild rats.

Infectious jaundice occurs sporadically in the United States. Since the *Spirochæta icterohæmorrhagiæ* is known to be widely distributed throughout America and may be transmitted to men, especially if they go barefoot, and work under unsanitary conditions in close proximity to rats, there can be little doubt that sporadic cases occur which are unrecognized. Light cases are difficult to differentiate from catarrhal jaundice, the suspicion of which may be excluded by the occurrence of severe invasion, high fever, prostration, congestion of the skin and mucous membranes, hemorrhages and intense albuminuria. The disease is not likely to spread or to become epidemic under normal sanitary conditions.

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CHAPTER VI

THE ROENTGEN RAY EXAMINATION OF THE GASTRO-INTESTINAL TRACT

BY. F. H. BAETJER, M.D.

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Since the discovery of the roentgen ray the diagnosis of gastro-intestinal diseases has undergone a marked revolution. This method of examination is probably the most valuable of all methods, but at the same time must not be looked upon as infallible. It is an extremely valuable and important link in the chain of evidence, but one must remember that it is simply one of many methods and its findings must be correlated with the clinical and other laboratory findings. While the roentgen examination may be looked upon as a method of precision, yet the percentage of errors is probably larger in gastro-intestinal examinations than in the examination of any other portion of the body. This is due to the fact that the stomach and intestines are subject to many variations and can be affected by many causes of either external or internal origin. The first important point to remember is that the gastro-intestinal tract is not a fixed anatomical part as are the bones, but that it is composed of organs which are in motion, constantly changing their shapes and which may be affected by mental conditions, drugs, food, etc., and that these organs are reacted upon reflexly by lesions in the abdomen not connected directly with the gastro-intestinal tract. It is the introduction of these variants, frequently of unknown origin, that leads one astray in interpretation. Then, too, there is no such thing as a fixed normal. The size, shape, position, motility, are all influenced by the habitus of the person. Every patient, therefore, is a new problem and one must construct a hypothetical normal for that individual and then consider whether there is a variation from this predetermined normal. When a diagnosis has been made, then the roentgenologist and clinician should discuss the case in detail and consider the roentgen findings in relation to the clinical and laboratory findings. Should the roentgen findings be completely at variance with the results obtained by all the other methods of examination, it is wise under such circumstances not to lay too much stress upon the roentgen results. When one considers, however, that this method of examination as applied to gastro-intestinal disease has been in use only a little over a decade, the results have been astonishing, and each year sees greater progress toward correctness of interpretation.

The roentgen examination should be made by an independent observer and not by the clinician. Otherwise, the case is approached with a subconscious prejudice towards that which has been found by the clinical methods.

The question is naturally asked: In what class of cases will the roentgen examination be of value? It should be used in all cases where cancer or ulcer are suspected; where there is a long history of digestive disturbance; and particularly in patients over forty years of age when cancer is so common and the only hope is in its early recognition.

The gastro-intestinal tract cannot be seen as such and so some shadow-casting substance must be introduced before the examination can be made. Barium or bismuth are the two best substances and they can be incorporated in fluids, cereals or mashed potatoes. When given by mouth, the esophagus, stomach, small intestines and the motility of the large bowel can be studied. For the detection of growths and strictures of the colon, barium or bismuth should be administered by means of an enema, as this completely fills the colon. The patient should be studied by the fluoroscope and plate methods, as one supplements the other.

If either the fluid or gruel type of meal is used, always adhere to it, as variations in the type of meal also cause variations in the activity of the stomach. One should also have a fixed time of administration, as the activity of the stomach varies as to whether the barium meal is given upon a fasting or full stomach. If the two points mentioned above are standardized, we have two constant factors to work with.

Esophagus.—This can best be studied by means of the fluoroscope during the administration of the barium meal. In a normal esophagus we have an approximately straight tube and, as the patient swallows, each individual bolus can be seen rapidly descending the esophagus and entering the stomach without any delay. Each bolus follows the other in orderly succession and equally spaced as to distance. When one finds that the individual swallows tend to run together, that generally means that there is some constriction and the point of slowing up should be carefully studied. In the normal esophagus there are two points where the lumen is slightly narrowed, one behind the sternoclavicular articulation and the other at the bifurcation of the trachea. Elsewhere the lumen is of the same size. This narrowing, however, is so slight that the speed of the descent of food is not affected in the least. When the passage of the food is delayed it is generally due to one of two causes, namely, spasm, or an actual stricture from a lesion within or pressure from a mass without. Spasms may be of two kinds. First, evanescent where there are short temporary spasms which vary as to position. In the same individual several may be seen occurring at different positions. They are of no significance, for there is no pathological basis for them.

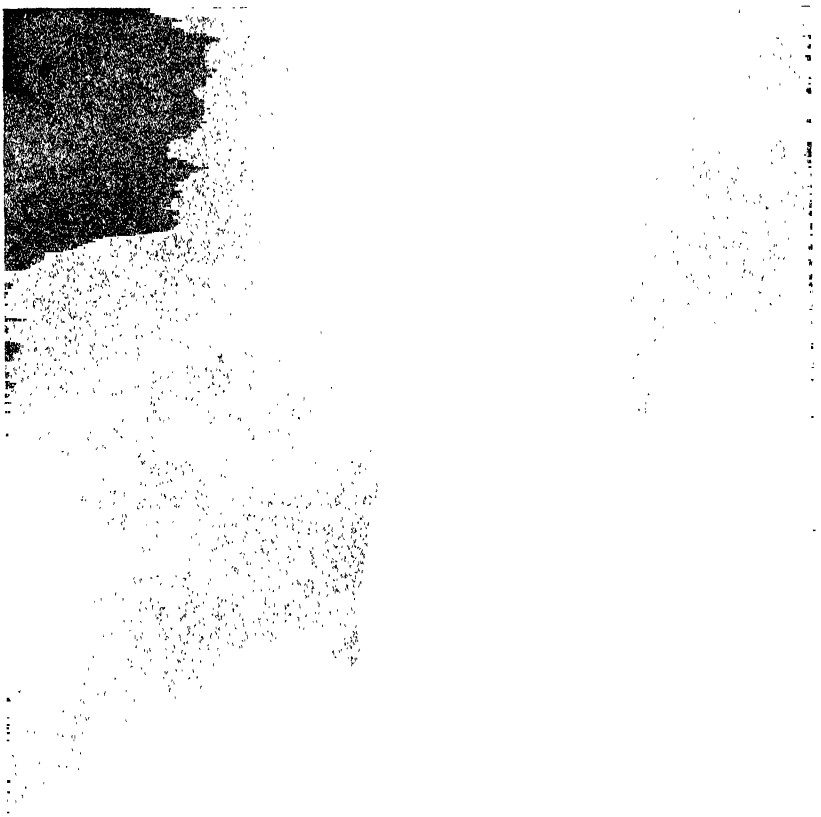

FIG. 1.—CARDIOSPASM WITH MARKED DILATATION OF THE ESOPHAGUS WITH THE "V" SHAPED OPENING IN THE MIDDLE OF THE LUMEN OF THE ESOPHAGUS.

Second, cardiospasm, which takes place at the cardiac end of the esophagus and is more or less permanent. This spasm is so persistent and so complete that it produces marked dilatation (Fig. 1). In fact there is no other lesson which produces as much dilatation as cardiospasm, and when advanced cases are seen the marked dilatation of the esophagus is not only a characteristic but an important diagnostic sign. If a clear view can be obtained at the point of spasm, one will see that the entire circumference is involved, so that the esophagus at that point is V-shaped, the apex pointing down and situated in what might be termed the middle of

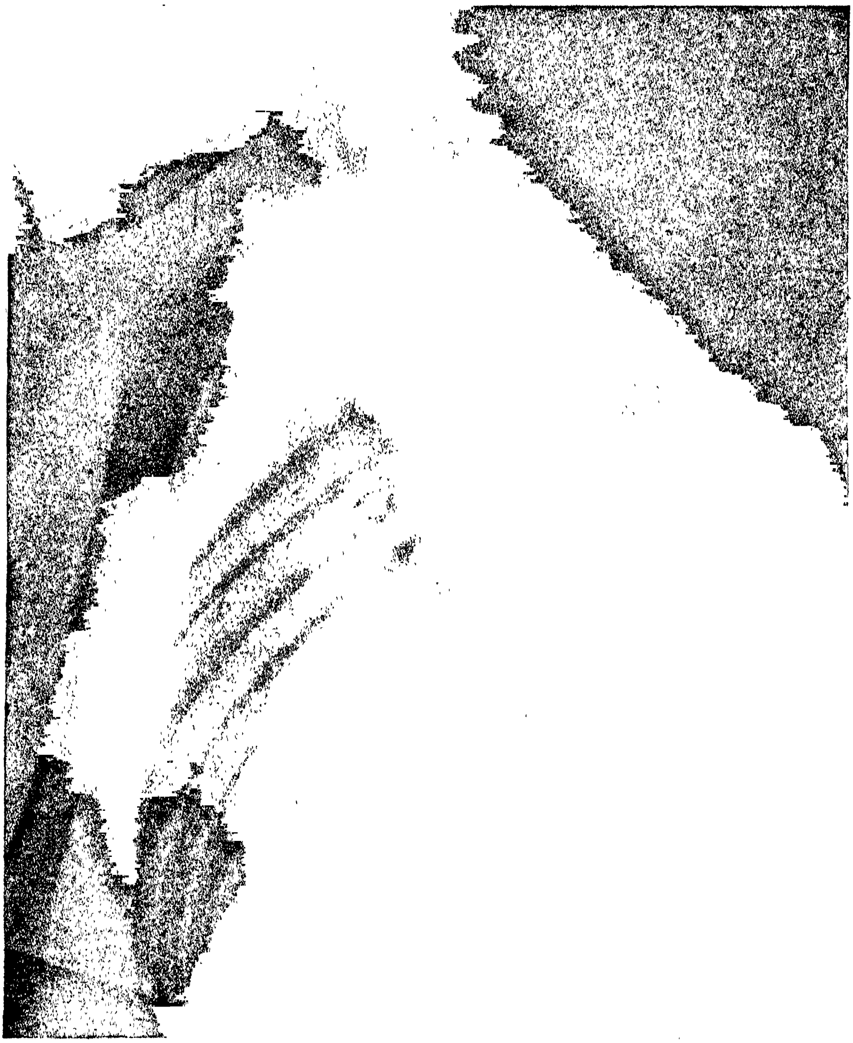

Fig. 2.—A Long Stricture in the Esophagus of a Child Due to the Accidental Drinking of a Washing Powder.

the lumen. On the other hand real strictures occur in one portion of the wall and, if a lumen is still present, it will be to one side. In such conditions it is quite striking to see the irregular barium-coated wall with the barium trickling through an opening on one side of the esophagus and not in the middle of the lumen as in spasm.

The age of the patient must always be considered, as in the very young real strictures are nearly always the result of burns due to the accidental drinking of lye or acid. These strictures are generally almost complete and may be several inches in length. The most common situ-

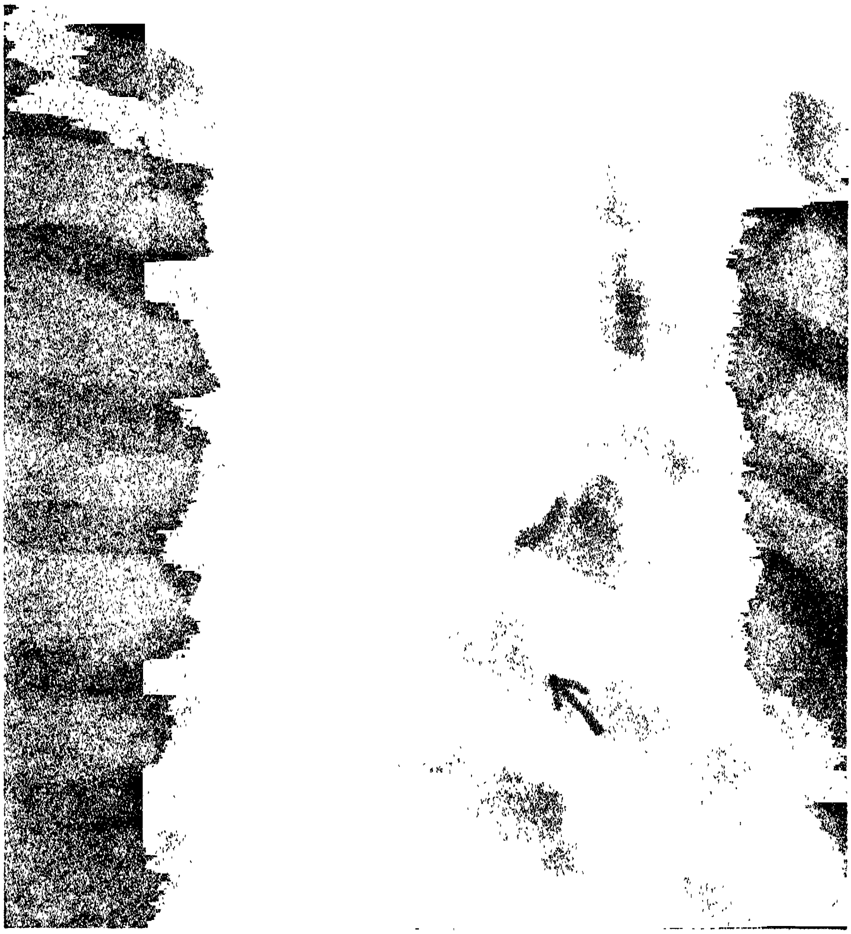

FIG. 3.—CARCINOMA OF THE MID-PORTION OF THE ESOPHAGUS WITH IRREGULAR, MOTH-EATEN WALLS AND THE OPENING ON ONE SIDE OF THE ESOPHAGUS INSTEAD OF THE MIDDLE OF THE LUMEN.

ation is approximately the mid-portion of the esophagus (Fig. 2). These strictures are seldom seen in adults, as in the young they either yield to treatment or the patient succumbs.

In middle and old age the stricture most commonly seen is that produced by carcinoma, although occasionally it may be the result of gumma. The lower and mid-portions of the esophagus, especially the cardiac end, seem to be the favorite sites for carcinoma. These strictures

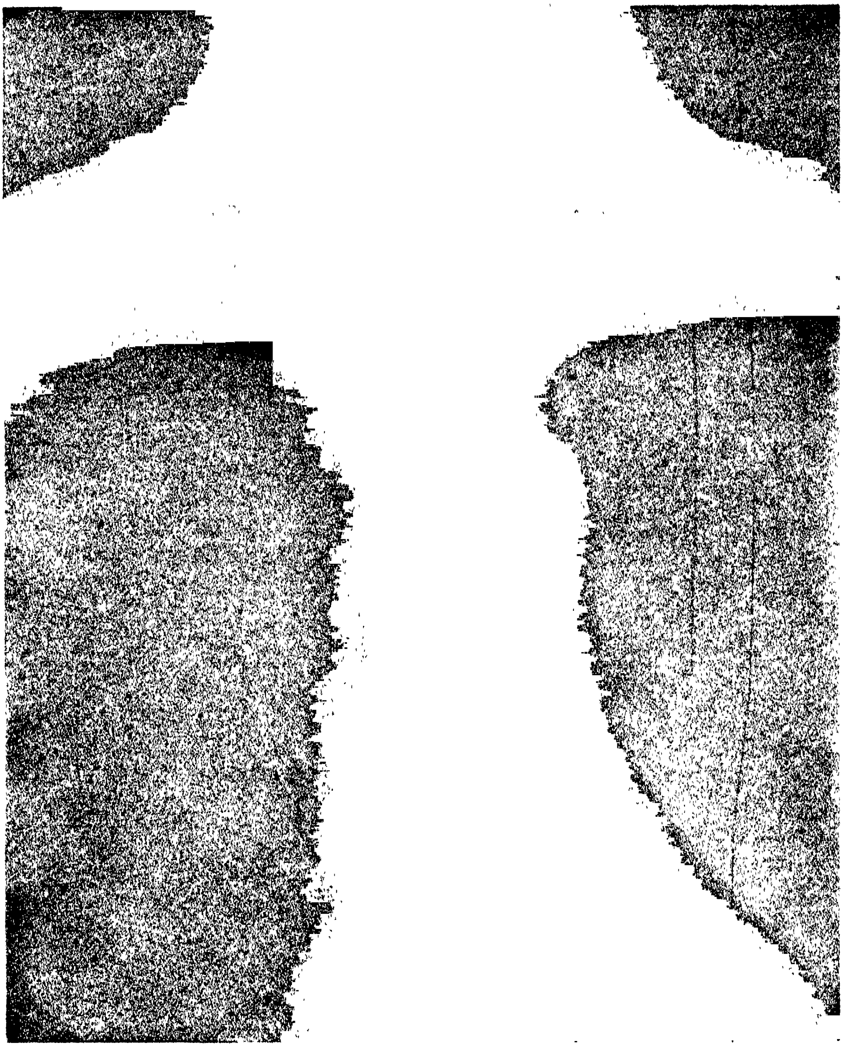

Fig. 4.—A Small Diverticulum Opposite the First Dorsal Vertebra. This is Lying to the Left of the Esophagus.

are generally incomplete. The walls in the region of the stricture are irregular and moth-eaten in appearance and the lumen has been encroached upon in some portion, so that the barium trickles through the stricture at one side (Fig. 3). There is generally some dilatation of the esophagus, but not the extreme degree that is seen with cardiospasm.

In the author's few cases of luetic strictures the appearance was the same as in malignancy, and the etiological diagnosis could not be made until the condition cleared up under antiluetic treatment. In several cases of large mediastinal growths the lumen of the esophagus was observed to be reduced in size, but the narrowed area was perfectly smooth and did not have the moth-eaten appearance of carcinoma.

Diverticula of the esophagus are most frequently seen in childhood. They are generally situated in the region of the sternoclavicular articulations and, by viewing the patient from various angles, a pouch-like structure can be seen lying to one side of the esophagus (Fig. 4). A diverticulum may be present and missed, as occasionally it may not fill with barium upon the first examination. If a diverticulm is suspected, repeated examinations should be made until either the pouch is filled or one can be sure none is present. The esophagus should also be examined from every angle, as the diverticulum may be lying directly behind the esophagus and not seen in the ordinary anteroposterior examination.

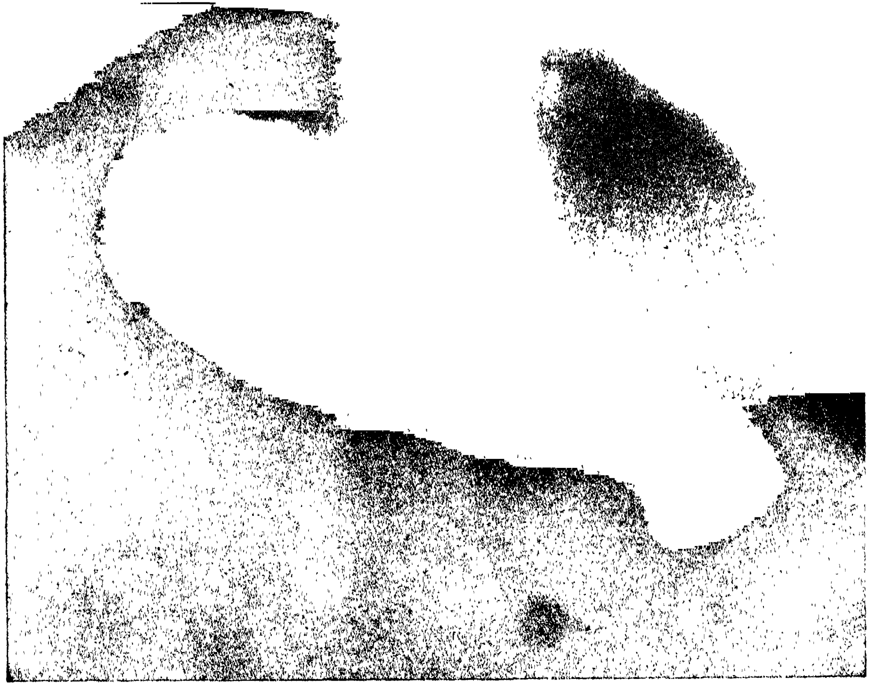

FIG. 5.—THE HIGH TRANSVERSE STOMACH SO FREQUENTLY SEEN IN VERY FAT INDIVIDUALS.

Stomach.—Before studying the pathological lesions it is well to consider the normal stomach. There is no such thing as a fixed normal stomach: it varies in size, shape, position, peristalsis, and motility. The habitus of the patient has a marked influence upon its position, shape and function. For the sake of classification the stomach can be considered as falling into three types both as to form and position: First, the high, tranverse type; second, the cow-horn type; and third, the fishhook or drain-trap type. The high, transverse type is seen in the fat patient

FIG. 6.—A COW-HORN STOMACH, THE PYLORUS BEING JUST A TRIFLE TO THE RIGHT OF THE MEDIAN LINE.

(Fig. 5). On account of the large amount of fat in the abdomen the stomach is pushed well up under the diaphram and the pylorus lies well to the right of the spine. In this type of stomach the evacuation time is relatively short, about three or four hours. This is caused by peristalsis and gravity working together.

The cow-horn type is seen in medium sized individuals (Fig. 6). It has a crescent shape, the fundus lying about the level of the umbilicus and the pylorus resting in the midline. Four or five hours is the normal time for evacuation.

Fig. 7.—A Fish-hook Stomach Lying Entirely to the Left of the Spine and the Fundus Well Down in the Pelvis.

The fishhook or drain-trap type is seen in thin individuals (Fig. 7). The fundus is generally found in the pelvis and the pylorus lies entirely to the left of the spine and below the umbilicus. Five to seven hours is the normal time for evacuation.

The time given for evacuation of the stomach is only approximate and may vary greatly, and yet be within normal range.

Peristalsis.—This is generally very smooth and regular, being most marked near the pylorus. It may be affected very markedly by the

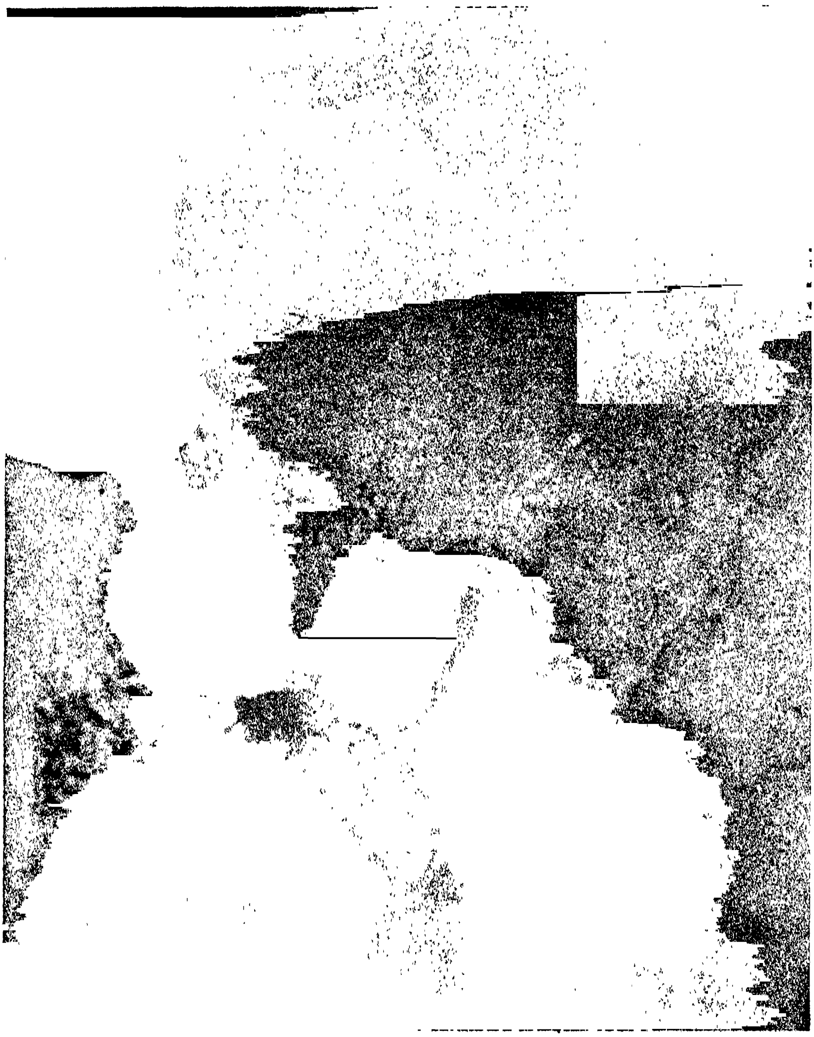

FIG. 8.—AN ULCER OF THE PYLORUS WITH THE BARIUM COMING OUT OF THE UPPER PART OF THE PYLORUS, THE ULCER PARTIALLY OBSTRUCTING THE LOWER PART. NOTE THE ACTIVE CONTRACTIONS, SO MARKED AS TO PRODUCE TYPICAL HOUR-GLASS FORMATION.

introduction of drugs and foods, and the mental attitude of the patient may for a time even inhibit its activity. It has been frequently observed in patients who express a deep distaste for the barium meal that the stomach will be perfectly inert and sluggish for a time. The peristaltic activity varies in different individuals and even in the same person. This difference in peristalsis we speak of as tone; it may vary from hypertonic to atonic.

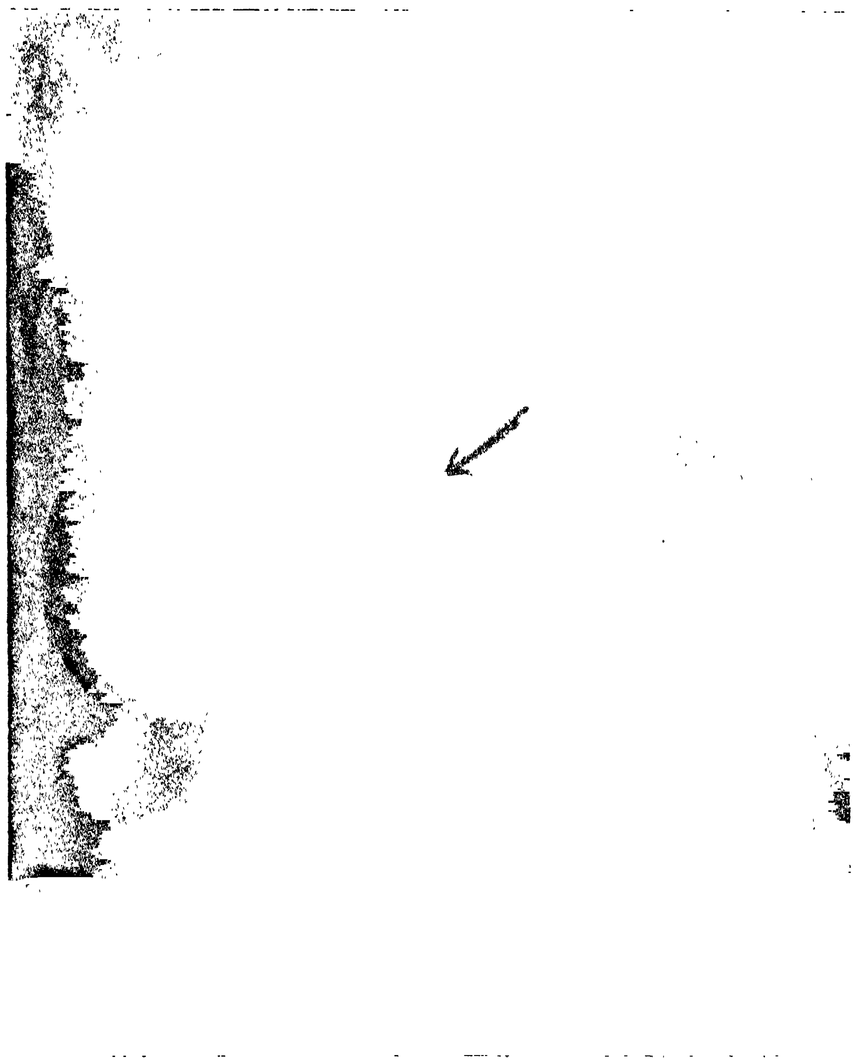

FIG. 9.—PENETRATING ULCER OF THE LESSER CURVATURE WITH THE POUCH FILLED WITH BARIUM. NOTE THE DEEP INCISURE ON THE GREATER CURVATURE OPPOSITE THE ULCER.

MOBILITY.—The stomach is fixed at the cardiac end and loosely fixed at the pyloric end, while the body of the stomach is freely movable under normal conditions.

LESIONS OF THE STOMACH.—The recognition of pathological changes in the stomach are dependent upon three things: first, changes in peristaltic activity; second, deformities of the walls of the stomach; third, interference with the emptying time. Let us now consider these changes in reference to specific lesions.

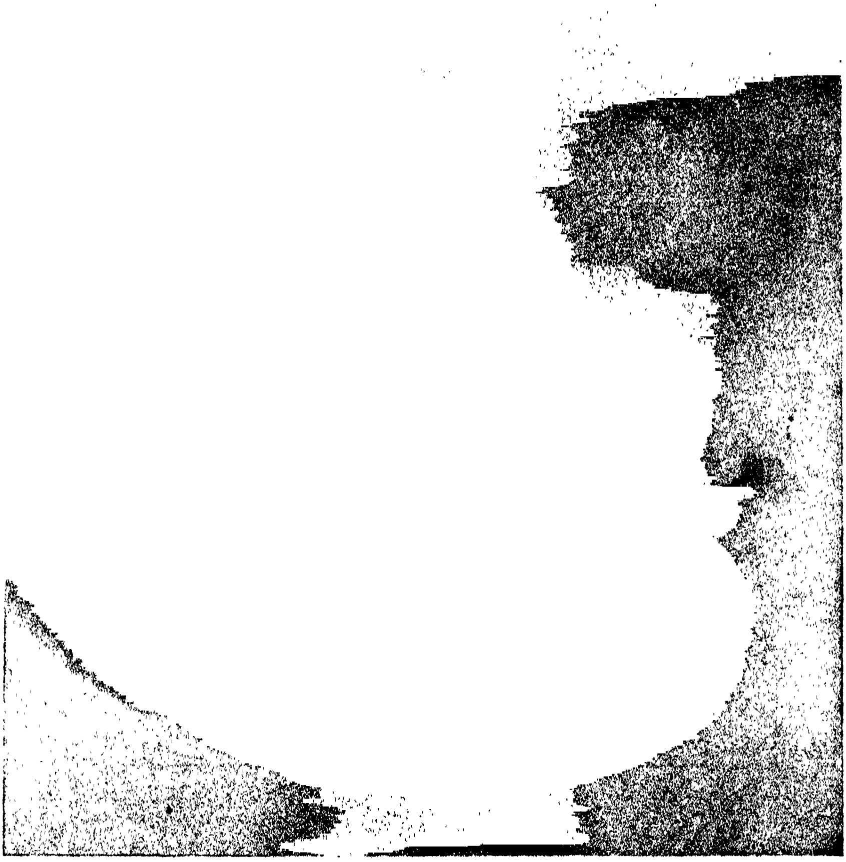

FIG. 10.—PYLORIC ULCER WITH OBSTRUCTION AND TWENTY-FOUR HOUR RETENTION.

ULCER.—These are most frequently seen in young and middle adult life. The lesion when found is generally situated at the pylorus or on the lesser curvature. While any portion of the lesser curvature may be involved, fully 90 per cent. of the ulcers are at the pylorus or on the first two inches of the lesser curvature (Fig. 8). Benign ulcers of the greater curvature are very rare. With acute ulcers there is marked hypertonicity with a spasm of the pylorus. The peristalsis may be so active that the stomach looks like a series of hourglasses. The peristaltic waves on the greater and lesser curvatures are uniform and equal in contraction except at the site of the ulcer. At that point the stomach

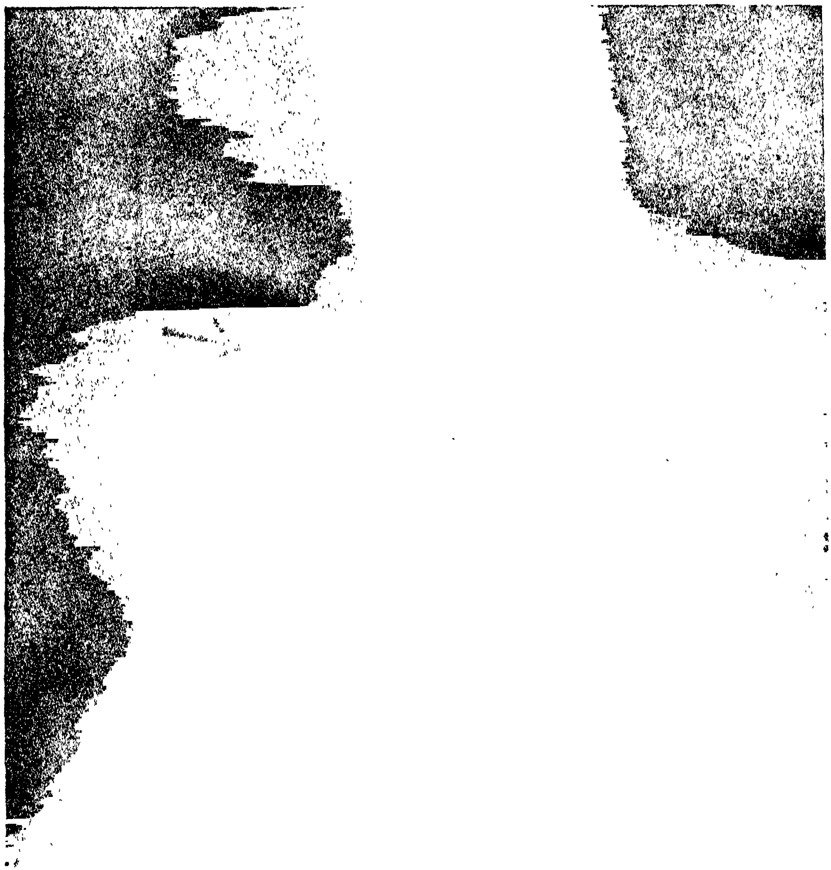

FIG. 11.—CARCINOMA OF THE CARDIAC END OF THE STOMACH, INVOLVING THE CARDIAC ORIFICE AND PRODUCING PARTIAL ESOPHAGEAL OBSTRUCTION.

wall is indurated and has lost its flexibility; consequently, there will be no peristaltic wave at that point. The wave on the greater curvature opposite the ulcer will be deeper to compensate for the absence of the wave on the lesser curvature where the ulcer is situated. This will produce a deep indentation on the greater curvature extending inward to the lesser curvature. This is spoken of as an incisura. It is frequently present, though it may be absent at times. The ulcer will show an interruption of the smooth continuity of the stomach wall and this is spoken of as a defect. These defects vary according to the extent and character of the ulcer. Spasm of the pylorus, which has already been mentioned, causes a marked delay in the evacuation time of the stomach.

The ulcer that gives the most striking picture is one of the penetrating type. This erodes one or more coats of the stomach wall, and the remaining coat or coats give way under peristaltic pressure and a pouch-like bag forms on the edge of the stomach (Fig. 9). This becomes filled with barium. When once seen it can always be recognized again. It occurs, however, in only one per cent. of the cases. This pouch is frequently surrounded by inflammatory tissue. Upon palpation a mass can be felt and clinically suggests a carcinoma. The roentgen picture, however, determines the real nature of the lesion.

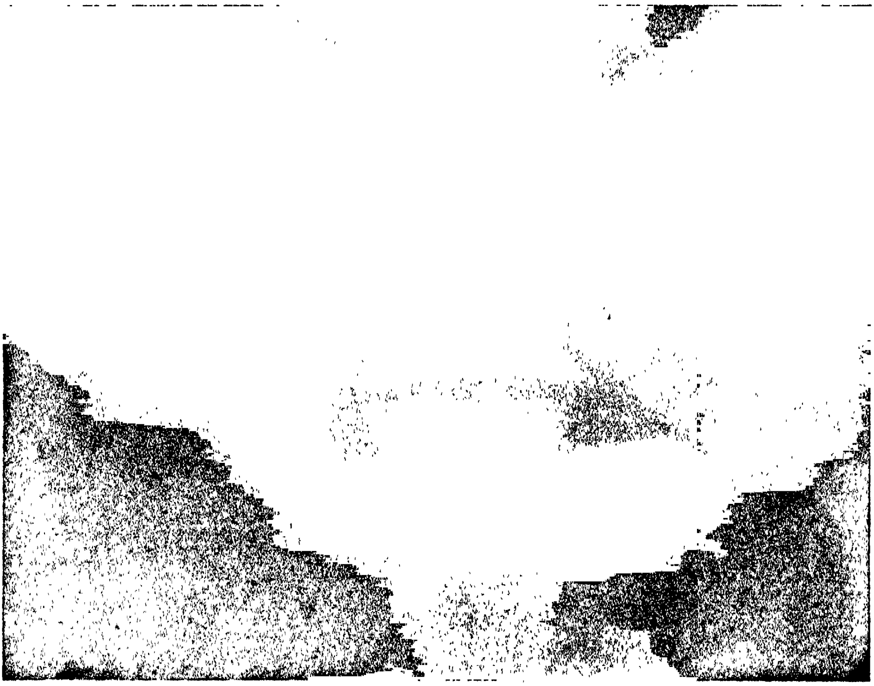

Fig. 12.—Large Carcinoma of the Greater and Lesser Curvature with the Pylorus Itself Intact and Consequently no Symptoms of Obstruction.

The main features of an ulcer on the lesser curvature are: first, increased peristaltic activity; second, the formation of an incisura; third, a filling defect; and fourth, delayed emptying time.

When the ulcer is situated at the pylorus it may produce more or less obstruction. The stomach then dilates, the pyloric antrum being the first part to show dilatation. When the dilatation becomes marked the stomach loses its tone and the peristaltic activity is markedly lessened, and in extreme cases may be almost absent. The emptying time may be extended to ten or twenty-four hours or more, depending upon the degree of obstruction (Fig. 10).

The above four signs are most frequently seen, though all of them may not be present in the same case. When adhesions occur arising from some condition outside of the stomach, as gall-bladder adhesions involving the pylorus, it is frequently very difficult and often impossible to determine whether the defect at the pylorus is due to ulcer or adhesions. The latter may act as a stimulus and there may be the same hypertonicity as seen with ulcer. After surgical interference, such as pyloroplasty or gastro-enterostomy, it is practically impossible to arrrive at any definite conclusions, as the size, shape, tone and motility of the stomach are completely altered.

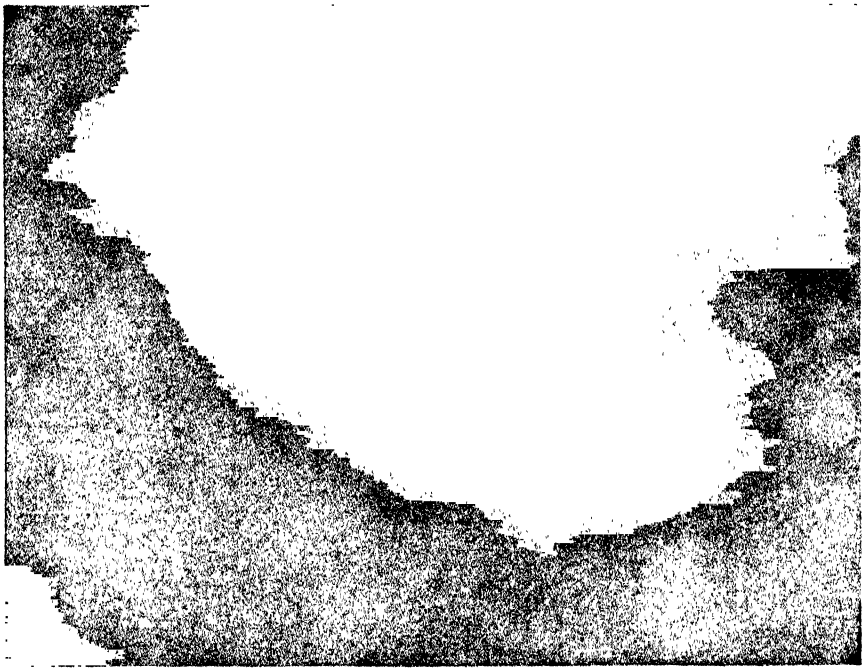

FIG. 13.—LARGE CARCINOMA INVOLVING THE ENTIRE STOMACH AND THE PYLORUS ITSELF IS A RIGID, FIXED OPENING WHICH CANNOT CONTRACT, SO THAT THE BARIUM FLOWS THROUGH FREELY. THIS PLATE WAS MADE THIRTY MINUTES AFTER THE ADMINISTRATION OF THE BARIUM AND THE STOMACH IS NOW PRACTICALLY EMPTY.

The question naturally arises as to how accurate is the roentgen diagnosis of ulcer. In the author's series of approximately 1,000 cases the error in diagnosis was between 20 and 25 per cent.

The greatest source of error was in interpreting deformities produced by adhesions as defects arising from ulcers. Sometimes the marked hypertonicity of the stomach may arise reflexly from gall-bladder adhesions or a chronic appendix, and there may be associated with it a pseudospastic defect that simulates an ulcer. In doubtful cases it is a good plan to administer atropin, and when the patient is thoroughly under the influence of the drug, then repeat the examination.

CARCINOMA.—For convenience of studying this condition it is well to divide the stomach into, first, the cardiac end; second, the body; third, the pyloric end. Carcinoma gives the same signs as does ulcer, that is, change in peristalsis, defects of the walls of the stomach and changes in the evacuation time, these changes, of course, depending upon the extent of the lesion. The site of the growth is the same as in ulcer, except that the greater curvature may be involved. It is the feeling of the author that very early carcinomatous lesions of the pylorus and lesser curvature cannot be differentiated from those of benign ulcers, while any lesion on the greater curvature is almost invariably malignant.

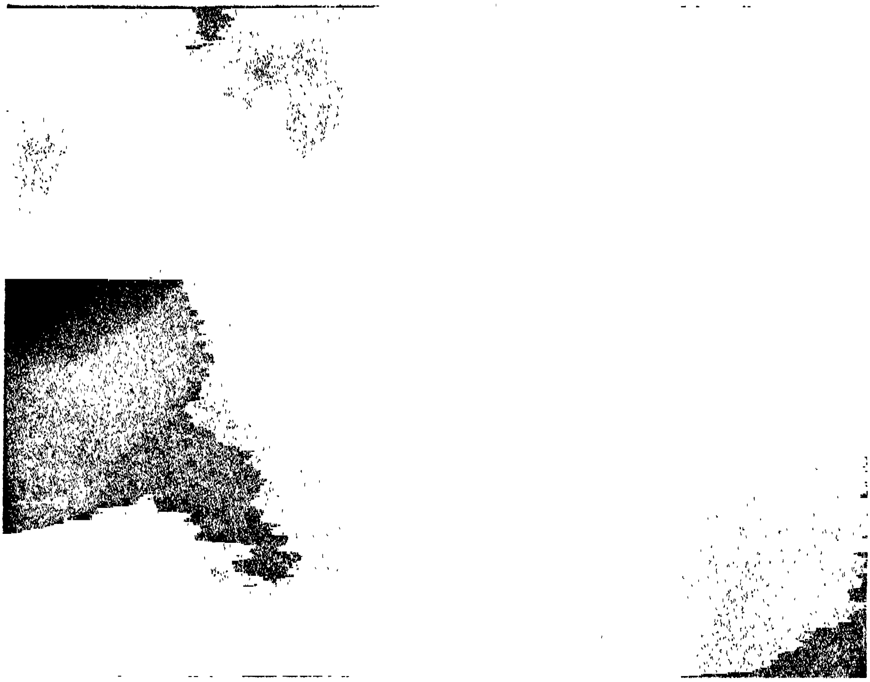

FIG. 14.—THE MARKED INDURATION OF THE STOMACH WALLS PREVENTED PERISTALTIC MOVEMENTS. THIS STOMACH DID NOT ALTER ITS SHAPE AT ALL DURING THE ENTIRE EXAMINATION.

Carcinoma of the cardiac end is generally seen quite early, as the cardiac opening is so frequently involved that symptons appear early and the patient presents himself for relief of symptoms (Fig. 11). In cases where the lesion is suspected in this portion of the stomach it is well to examine the patient lying upon the back, as in this position we get the complete filling of the cardiac end, thus doing away with the gastric bubble which is seen in the usual method of examination. The barium trickles slowly through the cardiac end and the stomach walls adjacent to it are irregular and moth-eaten. Occasionally a massive growth is seen in this area, but generally it is of the infiltrative kind.

In lesions in the body of the stomach one may have an extensive growth, and yet the patient may complain of very few symptoms, inasmuch as the motor activity is not interfered with (Fig. 12). As this lesion rarely affects the cardiac or pyloric orifices, obstructive symptoms are absent, the stomach emptying at the normal rate or perhaps a little more rapidly than normal (Fig. 13). For this reason this region is spoken of as "the silent area." The examination generally shows that the peristaltic waves are interrupted in their course at the site of the lesion, inasmuch as the lesion is hard and indurated and does not admit of further peristaltic movements (Fig. 14). In addition a persistent filling defect is almost always present. The filling defect is produced by infiltration of the walls or a massive growth in the stomach wall bulging generally more or less into the lumen of the organ itself, so that it is outlined by the barium in the stomach proper and presents the appearance of an indentation (Fig. 15).

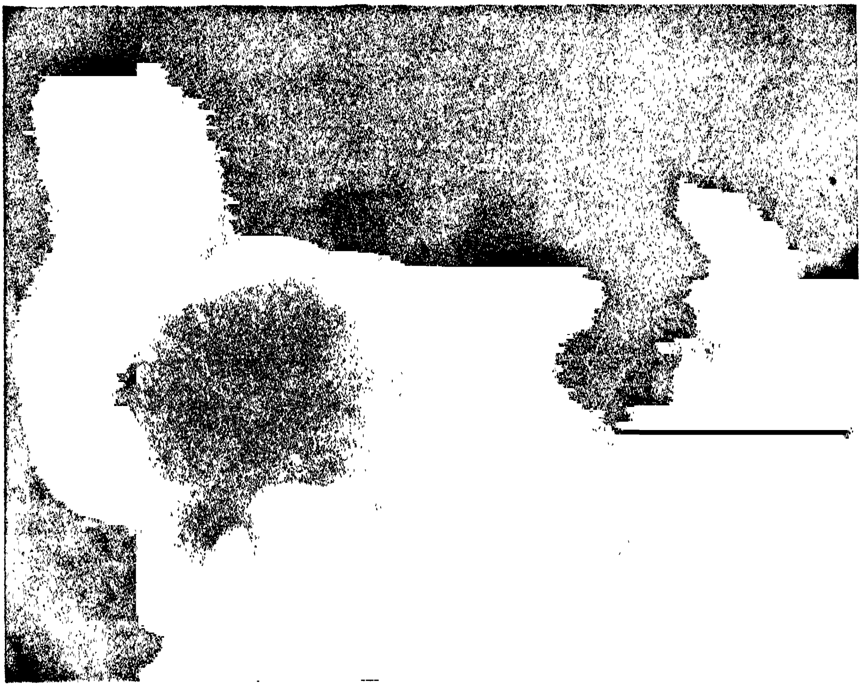

FIG. 15.—LARGE CARCINOMA OF THE GREATER CURVATURE EXTENDING INWARD AND ALMOST FILLING THE LUMEN OF THE STOMACH.

In the third class of cases, that is, in lesions around the pylorus, the symptoms arise early as a rule, and are due to a partial obstruction which quickly attracts the patient's attention to the gastric disorder (Fig. 16).

We may classify growths at the pylorus into two forms. The first, the annular carcinoma, has its origin within the pylorus itself, and although it may be very small in size, produces symptoms relatively early, as it soon causes a greater or less degree of obstruction (Fig. 17). Occasionally such a growth will completely infiltrate the pyloric ring without obstructing the lumen and, consequently, we have a rigidly fixed open pylorus through which the barium pours, quickly emptying the stomach (Fig. 18).

The second type also has its origin at the plyorus; it is not annular in form, but invasive, most frequently extending along the wall of the stomach. Such a growth may or may not produce obstruction (Fig. 19).

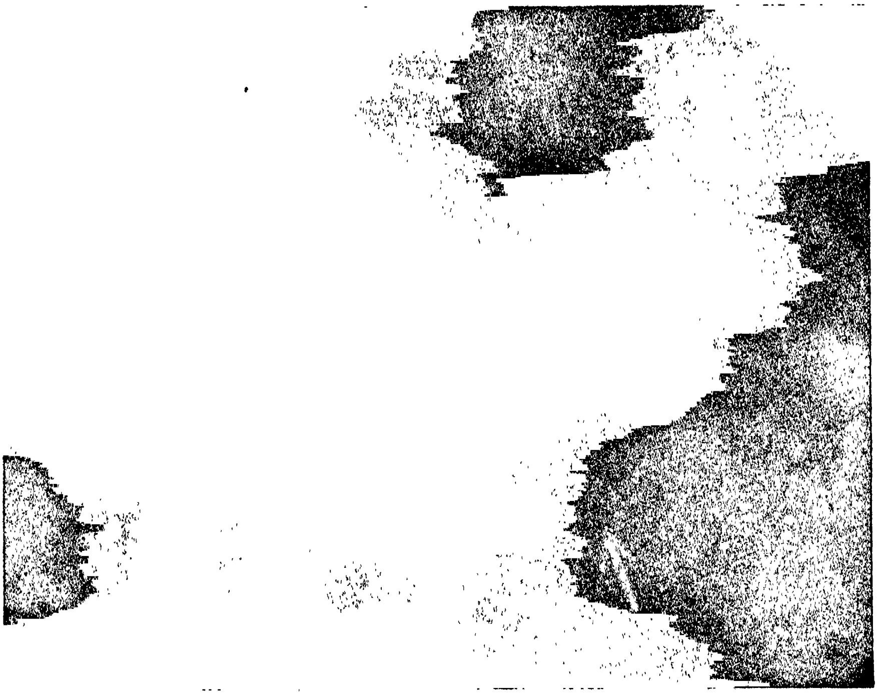

FIG. 16.—CARCINOMA OF THE PYLORUS PRODUCING OBSTRUCTION.

The annular type of carcinoma is easily recognized inasmuch as the stomach is observed to be perfectly normal except at the pyloric end. The pylorus, instead of being clear-cut and sharp, is generally slightly depressed in the center and somewhat thickened, so that the structure assumes the shape of a crater.

The second type of carcinoma may exist for some time without being recognized, because in the absence of any symptoms of obstruction, the patient being still in a comfortable state, does not present himself for an examination. In this type of growth one finds a persistent filling defect in or close to the pylorus and ascending away from it. The mass is indurated and free from peristaltic waves since the waves pass over the area and are lost.

In these cases of partial obstruction the stomach begins to show a very definite change. In the early stages we find active contractions of the organ with a slow elimination of its contents. Another very significant sign is that a portion of the stomach just within the pylorus and on the greater curvature in the pyloric region shows a tendency to bulge. The cause of this condition is as follows: The active contractions of the stomach force all of the food toward the pyloric region; the pylorus not being patent, as a result of this constant pressure, the prepyloric region becomes dilated, so that the plate presents the pylorus apparently not at the end of the stomach, but with the prepyloric region extending further to the right than the pylorus, the latter resting on top of the stomach and pointing to the splenic region. This prepyloric bulging is dependent largely upon the duration of the affection (Fig. 20). In the early stages it is very small, but as the condition advances, it may reach the size of a hen's egg. As the condition advances still further, dilatation begins to take place, and after a time practically the entire fundus yields, so that a typical saccular formation is produced and all the barium rests in the fundus. When the stomach is in this condition, there will be a retention of contents from ten to twenty-four hours or more (Fig. 21).

FIG. 17.—CARCINOMA OF THE PYLORUS PRODUCING OBSTRUCTION, AND THE GROWTH IS BEGINNING TO EXTEND UPON THE LESSER CURVATURE.

Occasionally a growth external to the stomach may be so situated that the mass will press upon the stomach wall and produce a large indentation simulating a defect. Upon palpation, however, the stomach can be moved and the defect will disappear (Fig. 22).

The author has seen one case where a large, smooth defect was present upon the lesser curvature close to the pylorus producing some obstruction. The stomach was firmly fixed and could not be moved. The gastric analysis showed absence of hydrochloric acid. A diagnosis of carcinoma was made, but an exploratory operation showed this defect to be caused by an abnormal lobe of the liver adherent to the stomach and impinging upon the gastric lumen.

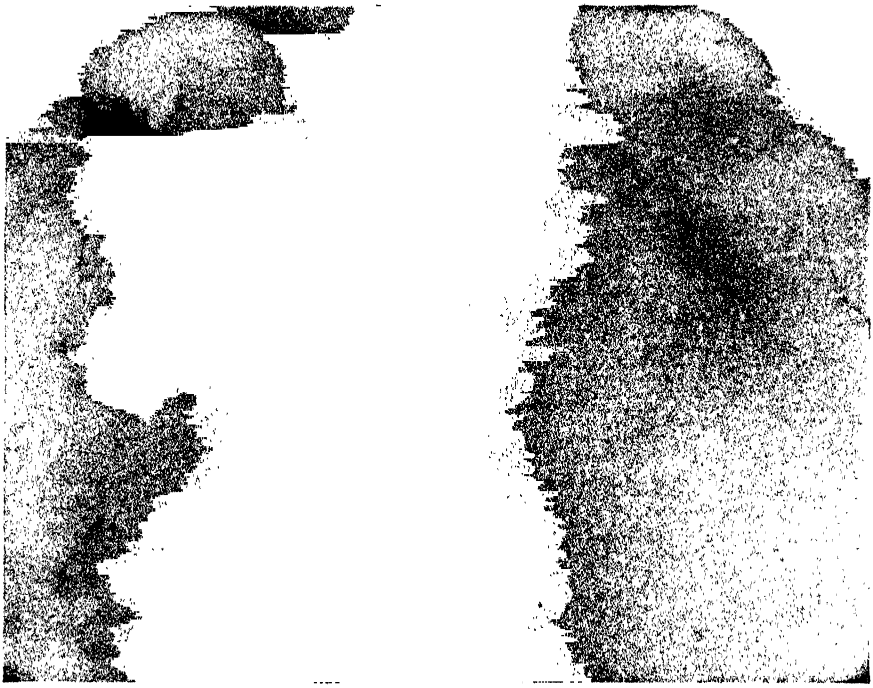

Fig. 18.—Extensive Carcinoma of the Stomach with the Pyloric End Changed into a Tube-like Structure, but not Producing any Obstruction.

Unless due care be exercised one may easily be misled by certain reflex or spastic conditions of the stomach. It is not uncommon to observe a stomach with a filling defect due to spasm which will continue for a period of an hour or two. In one case under observation a similar filling defect due to spasm lasted for about forty-eight hours and disappeared only when atropin was administered. In doubtful cases the question of spasm of the stomach may be eliminated by the administration of full doses of atropin for one or two days until the patient is well under its influence. A second examination made under these conditions will immediately show that the suspicious area has completely vanished, and that the condition previously observed was due to spasm.

In the study of gastric carcinoma the differential diagnosis between this condition and ulcer is often very difficult. It is still a much debated question as to whether carcinoma of the stomach has its origin primarily as such, or is the result of a transition from an old ulcer. If the latter view be correct, one can readily understand the difficulty in determining when the benign condition begins its transition into malignancy. The situation of both carcinoma and ulcer is frequently the same, with the exception of the greater curvature, where carcinoma seems to occur and never ulcer. In the differential diagnosis between the two conditions the points to be taken into consideration are as follows:

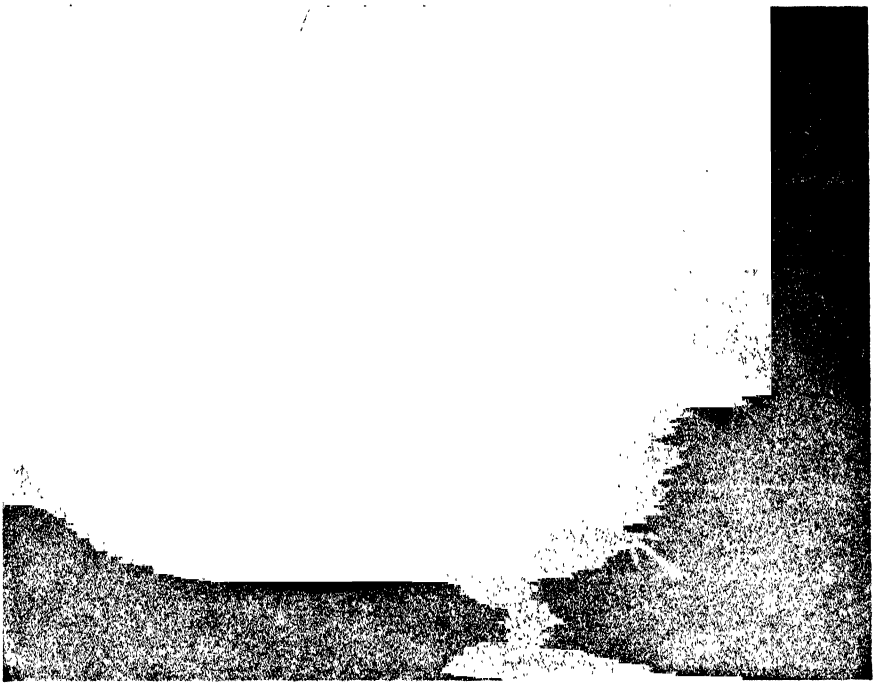

Fig. 19.—Annular Carcinoma of the Pylorus with Obstruction.

First, in carcinoma, unless there is obstruction, there is nearly always active peristalsis with rapid evacuation of contents. In ulcer there is hyperperistalsis and slow evacuation of the gastric contents.

Second, carcinoma may occur in any part of the stomach. The invasive lesions are more frequently seen on the lesser curvature near the pylorus, and less frequently on the greater curvature, whereas ulcer is generally observed on the lesser curvature near the pylorus and is practically never seen on the greater curvature.

Filling Defects.—In carcinoma the filling defect is generally surrounded by an invasive area which interferes with peristalsis, producing an apparently large dead area. In ulcer the defect is smaller and is not so apt to have the immediate peristaltic wave interfered with, although occasionally if the ulcer is a large indurated one the same peristaltic changes will be noted.

Carcinoma of the pylorus in the earliest stages is generally annular and produces a crater-like appearance, while ulcer in that area gives an irregular filling defect. When any of these conditions pass on to the obstructive stage, the change that is caused by the dilatation may mask the signs associated with the filling defect.

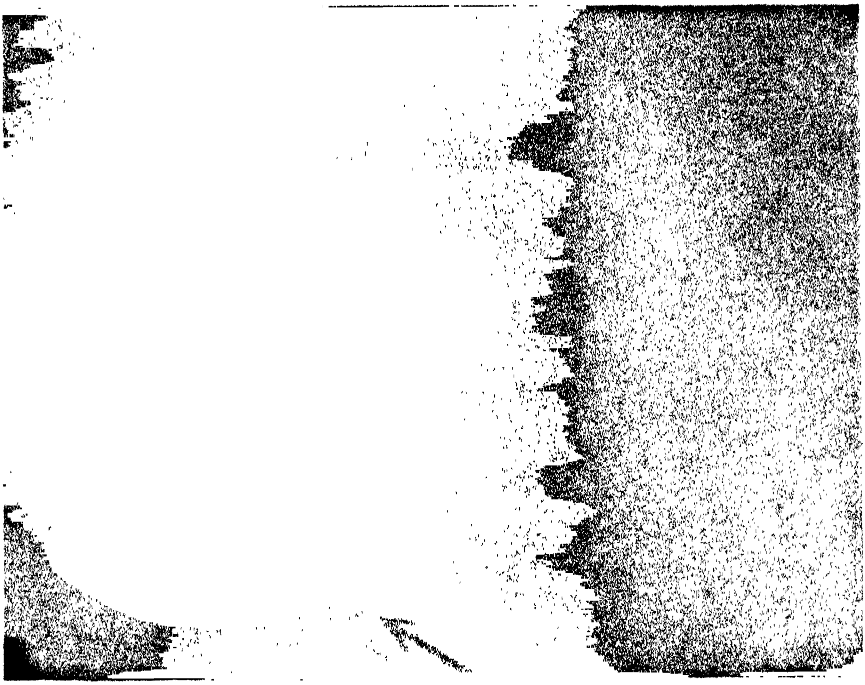

FIG. 20.—CARCINOMA OF THE PYLORUS WITH MARKED PREPYLORIC BULGING.

In the author's experience in the very early stages of gastric cancer, it is frequently impossible to determine whether we are dealing with a malignant or benign ulcerative lesion. The main object, however, is to determine whether the lesion at hand is really an ulcer or not. Inasmuch as indurated gastric ulcers have at times a tendency to become malignant and produce roentgenograms similar to those which are cancerous, they must be included in the same class. In the later stages of gastric cancer, however, when the growth is large it is usually a very simple matter to arrive at correct conclusions regarding its malignancy, but at this stage while the clinician is not dependent upon the x-ray for a diagnosis (the symptoms being well established), yet it furnishes important evidence as to whether the tumor is operable or not; it establishes clearly the location and extent and also the degree of obstruction.

Syphilis of the stomach is a very uncommon lesion. It gives practically the same signs as carcinoma. A large filling defect will be present, the walls will be irregular and worm-eaten and there may be hour-glass contractions (Fig. 23). The author feels that it is impossible to distinguish this lesion from one produced by cancer. A positive Wassermann may be helpful, but it is not conclusive, as cancer is frequently seen in syphilitics. When one sees a young individual with a large lesion of the stomach and yet showing no signs of a serious constitutional disease, one must have syphilis in mind. In such cases, when antiluetic treatment is administered, and the lesion completely disappears, the presumptive evidence is strongly in favor of the lesion having been syphilitic.

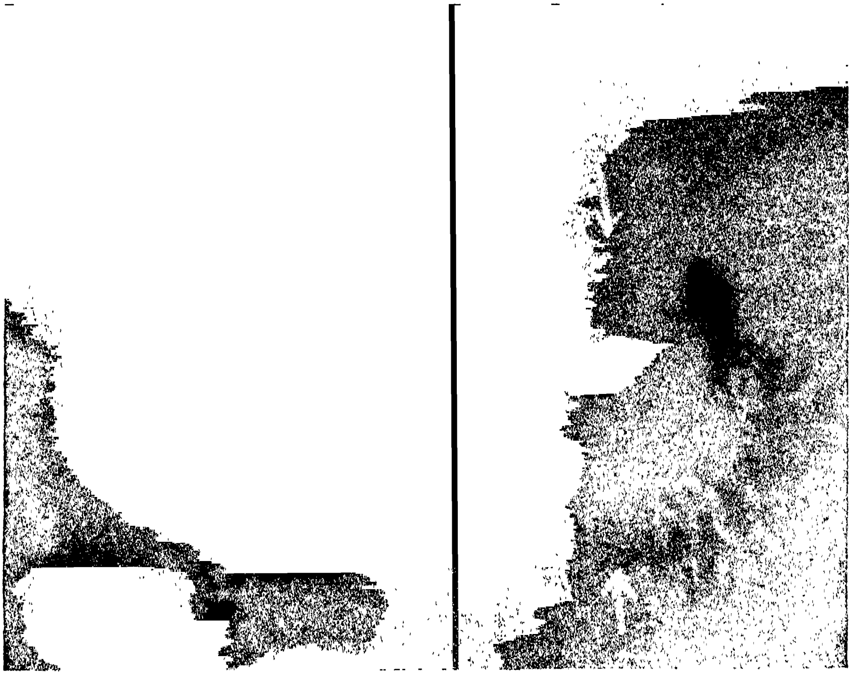

FIG. 21.—CARCINOMA OF THE PYLORUS WITH TWENTY-FOUR HOUR RETENTION.

It is well to bear in mind, however, that carcinoma is occasionally seen in the young and one must not make a diagnosis of lues simply because the patient is quite young and has a positive Wassermann. The author has had just such a case in a man of twenty-three, where the lesion was malignant and not luetic.

Besides the lesions already mentioned abnormalities can be determined, transposition of the viscera being the most common. Occasionally one will find a hernia of the stomach into the thorax (Fig. 24).

The roentgen examination is of great assistance in the detection of foreign bodies. The author has observed pins, nails, hair-balls, finger

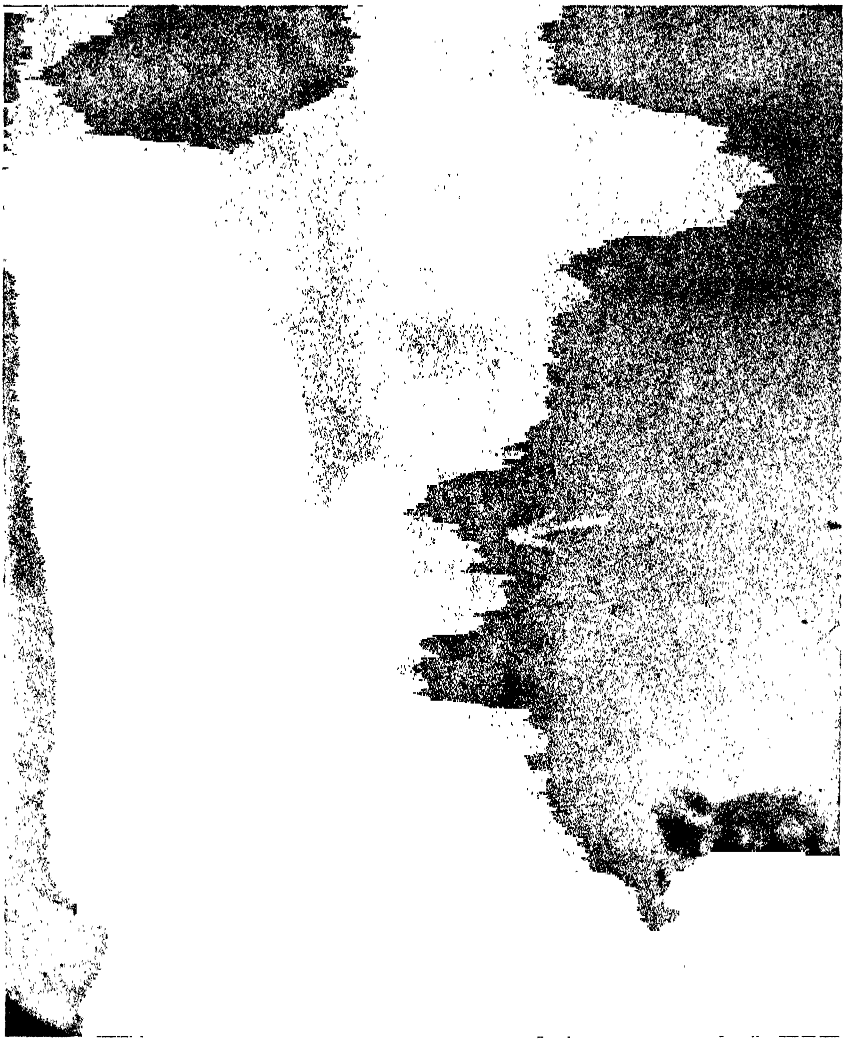

FIG. 22.—A MASS IN THE OMENTUM PRESSING UPON THE PYLORUS AND GREATER CURVATURE PRODUCING A DEFORMITY. UPON PALPATION THE MASS COULD BE PUSHED AWAY AND THE STOMACH ASSUMED A NORMAL APPEARANCE.

rings, portions of stomach tubes, glass, false teeth, etc., in the stomach, and on one occasion a small dog chain nearly six feet in length was found.

Examination of the stomach after medical treatment of an ulcer is of great assistance in determining the extent of healing. The gradual disappearance of the defect can be observed. After the defect has disappeared peristalsis should return to normal. If marked hyperperistalsis still persists, although the filling defect has disappeared, the ulcer has not completely healed. It has been the author's experience that if a patient goes on an unrestricted diet at that time there will be a recurrence of the symptoms.

The proper functioning of a gastro-enterostomy can be readily determined. In some cases where there is a partial obstruction and a gastro-enterostomy has been done, there may be a return of the symptoms. In such cases a partial closing of the gastro-enterostomy opening has been observed (Fig. 25).

In resection of the stomach for growths it is often impossible to determine whether there is a return of the disease. The operation has so completely changed the shape and activity of the organ that it is difficult to determine whether the altered shape and activity are due to surgical interference or disease.

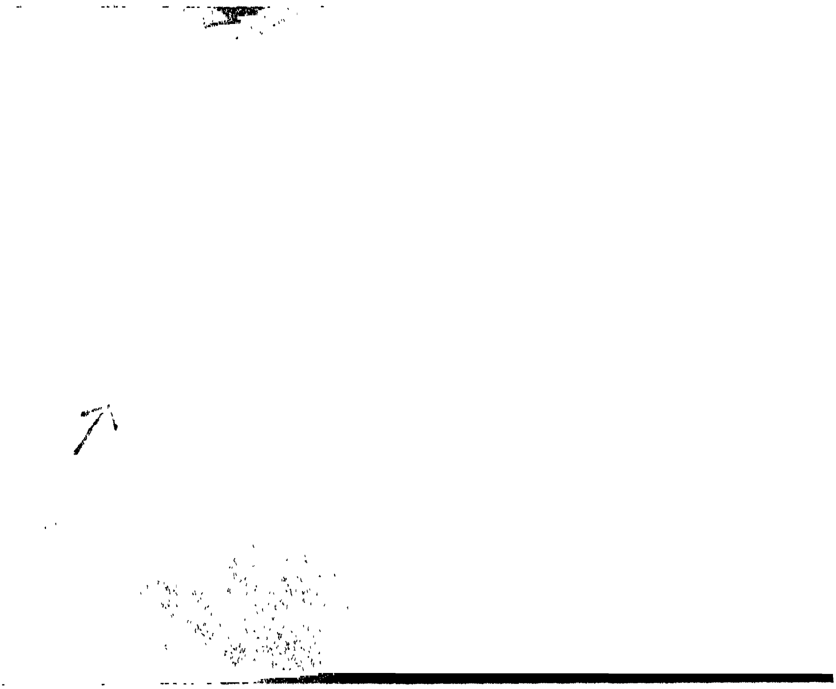

FIG. 23.—LUETIC STRICTURE WHICH WAS THOUGHT TO BE CARCINOMA, BUT AT OPERATION WAS FOUND TO BE LUETIC.

Small Intestines.—Of all the lesions in the small intestines, duodenal ulcer is probably the most common (Fig. 26). The first portion of the duodenum is the part most generally affected. Just as in gastric ulcer the two cardinal signs are changes in the peristaltic activity of the stomach and duodenum, and filling defects. The first part of the duodenum is called the "cap" and morphologically it is very similar to the stomach. Any stimulation of the cap reflexly stimulates the stomach and peristaltic activity is set up, but with a marked difference as to motility. With gastric ulcers there is increased peristaltic activity

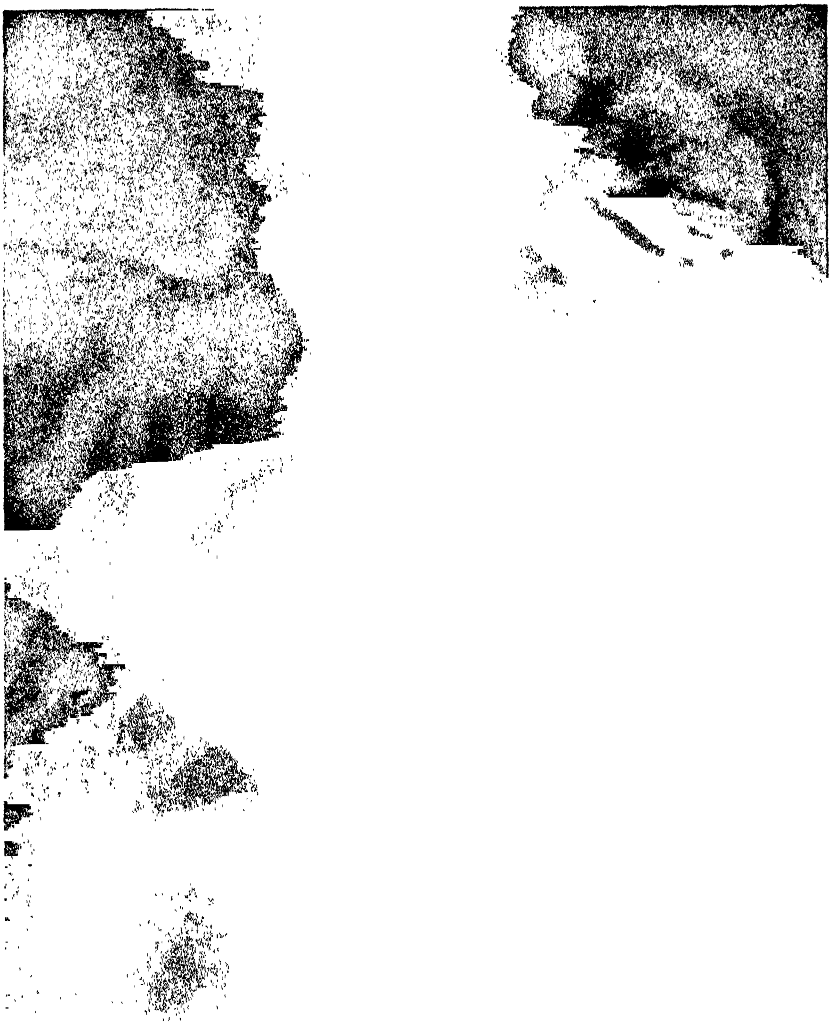

FIG. 24.—HERNIA OF THE STOMACH INTO THE RIGHT THORAX. NOTE THE BARIUM-COATED STRING WHICH THE PATIENT HAD SWALLOWED.

associated with spasm of the pylorus with resulting delayed motility. In duodenal ulcers, when food touches the raw ulcerative surface, the duodenum contracts and pushes on the food, but the contraction of the duodenum sets up a peristaltic wave in the stomach and this in turn pours fresh material into the duodenum, which in turn contracts again; consequently the harder the duodenum contracts just that much more the stomach contracts. There is no valve in the duodenum that can go into spasm and bring about a general spasm of the duodenum and stomach, producing a temporary arrest of activity as in gastric ulcer. On

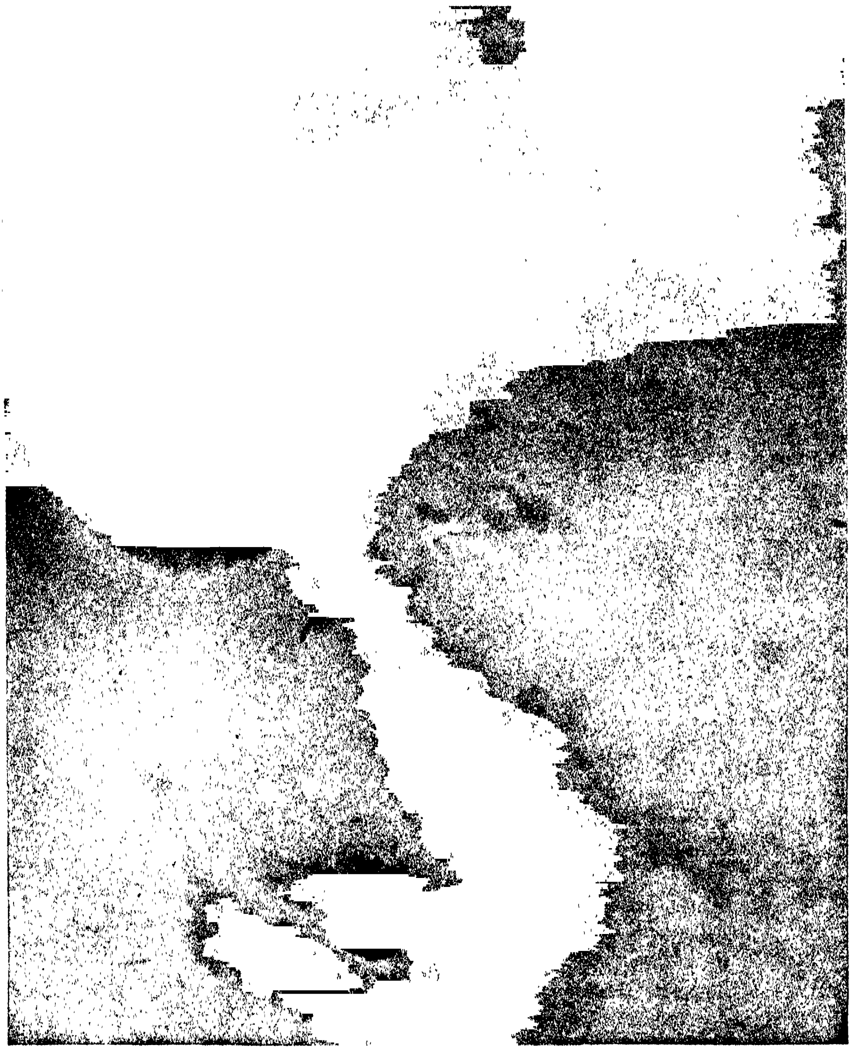

FIG. 25.—CARCINOMA OF THE PYLORUS PRODUCING ALMOST COMPLETE OBSTRUCTION. GASTRO-ENTEROSTOMY OPENING WAS DONE AND SIX MONTHS LATER THE CARCINOMA BEGAN TO INVOLVE THE NEW OPENING PRODUCING OBSTRUCTION. THIS EXAMINATION WAS MADE FIVE HOURS AFTER THE ADMINISTRATION OF THE BARIUM.

the contrary the entire functions of the stomach and duodenum are speeded up and have increased peristaltic activity with rapid emptying of the stomach. The cardinal difference then is that in gastric ulcer we have hyperperistalsis with delayed motility, while in duodenal ulcer we have hyperperistalsis with hypermotility. In simple uncomplicated cases of duodenal ulcers it is quite common to see the stomach emptied in fifteen minutes. In cases, however, where there are adhesions or slight obstructions there may be six-hour residue in the stomach.

Filling defects are much more definite in the duodenum than in the stomach. In fact if a normal duodenal cap can be shown we can definitely rule out ulcer. Adhesions from the gall-bladder may produce defects that closely simulate those of ulcer. Upper right quadrant adhesions may be so extensive as to completely mask the primary lesion. The author has seen a number of cases explored where in one set of cases the primary lesion was an ulcer, and in the second set of cases gall-bladder infection was the cause of the adhesions. From an x-ray standpoint it was impossible to differentiate these conditions. Primary carcinoma of the duodenal cap is so rare that it need only be mentioned. Only one case was seen by the author and it is needless to add that the correct diagnosis was made only at autopsy.

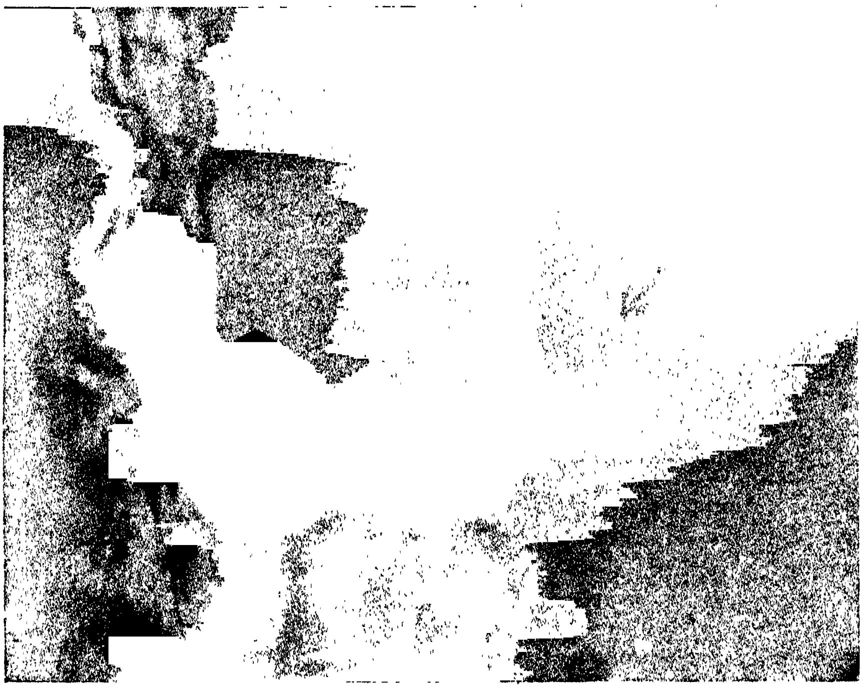

FIG. 26.—LARGE DUODENAL ULCER PRODUCING MARKED DEFORMITY OF THE CAP.

Ulcers of the second and third portions of the duodenum are rare, but if obstruction is present there may be dilatation behind the obstruction. Several cases of large dilatation of the duodenum have been noted and yet at operation no cause could be found (Fig. 27). These must be looked upon as idiopathic dilatations and may be the result of some nerve inhibition.

Diverticula of the duodenum are occasionally seen. They are readily recognized as solid masses of barium just to one side of the duodenum and the pouches vary in size from a dime to a quarter, and remain filled with barium even after the stomach and duodenum are empty. They have been observed to remain filled for forty-eight hours (Fig. 28). Just as in esophagus diverticula they may be present, but the lumen of the pouch may not always be patent and so they may not be detected.

The duodenal cap is the only portion of the small intestines that is sharply defined under normal conditions. The rapid passage of the contents does not completely fill the intestines and consequently the small intestines are not shown in detail. A plate shows irregular feathery masses scattered throughout the area where the intestines lie. Whenever the small intestines are well filled and clearly outlined, we are dealing either with spasm, delayed motility from adhesions, or obstruction from growths. It is quite difficult to determine sometimes what causes the obstructive condition. When the small intestines are all matted together it is generally the result of some chronic inflammatory condition, and tuberculous peritonitis is the most common cause. In upper and lower right quadrant adhesions the small intestines may be massed together in either of these quadrants according to that which is affected. In large tumors of the gall-bladder or pelvic region the small intestines may be markedly displaced, thus giving a clue to the condition.

Fig. 27.—Idiopathic Dilatation of the Duodenum. No Cause for this Could Be Found at Operation.

The Colon.—In the examination of this organ the barium meal as well

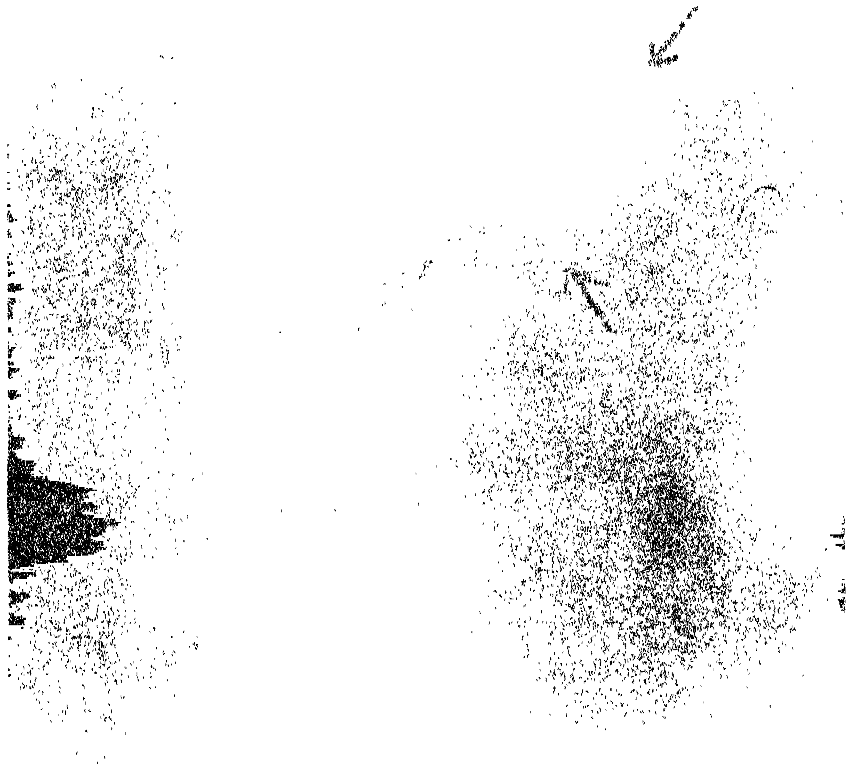

FIG. 28.—DIVERTICULUM OF THE DUODENUM TWENTY-FOUR HOURS AFTER THE ADMINISTRATION OF THE BARIUM MEAL.

as the enema are used. The latter half of the colon is best demonstrated by means of the enema, and it is the only method by which complete filling can be obtained. The colon varies in size and position according to the habitus of the patient. It varies its position just as the stomach does. We find the high colon in fat individuals and the pelvic colon in the very thin. In studying the colon stress should be laid upon its activity and not its position.

The normal colon varies as to size, being largest at the cecum, gradually narrowing until the rectum is reached, and again widening at the

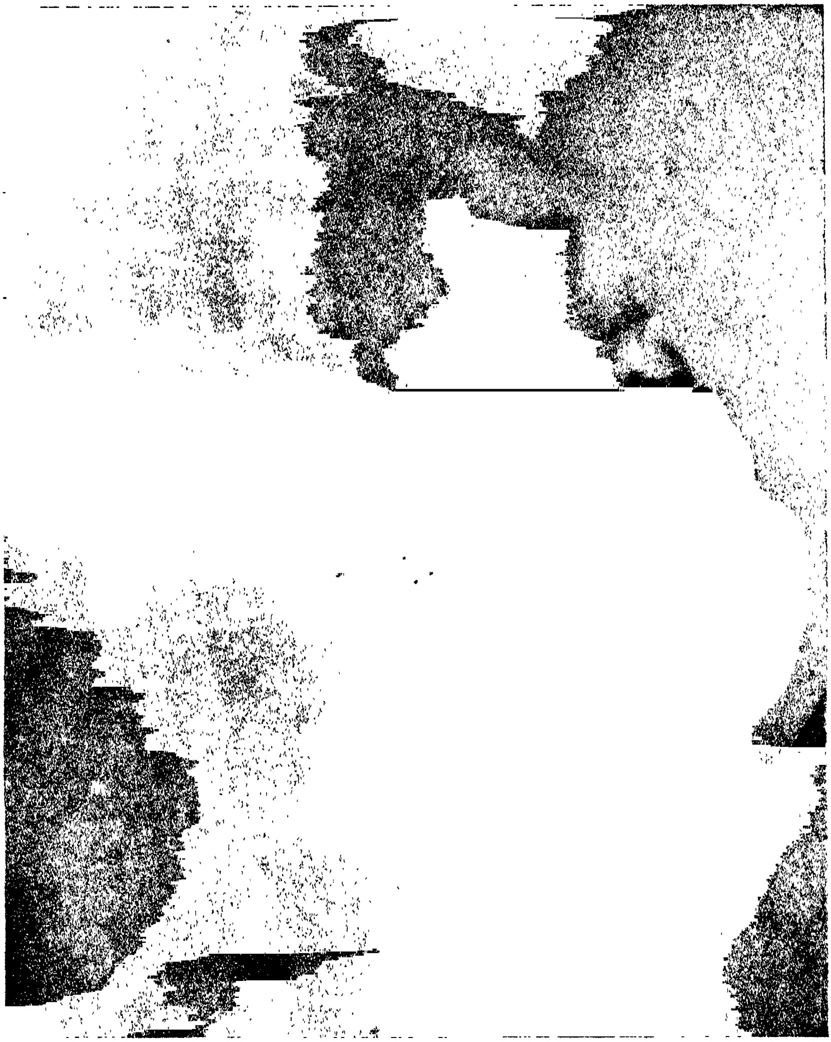

FIG. 29.—RETROCECAL APPENDIX, TIP OF IT BEING SEEN ON THE INNER SIDE OF THE CECUM AND ADHERENT.

rectal ampulla. While the passage of contents through the small intestine is rapid, the large bowel acts as a reservoir and the movement is relatively slow. In a normal individual the meal should be in the cecum two hours after the stomach has completely emptied. The head of the meal should then be at the hepatic flexure in from six to nine hours, the splenic flexure nine to twelve hours, and in the rectum from sixteen to twenty-four hours. The stomach and small intestines are constantly in motion during a meal. The movements in the colon are "mass movements" with fairly long intervals of complete rest.

FIG. 30.—APPENDIX FILLED WITH BARIUM, KINKED AND ADHERENT TO THE CECUM.

In studying the colon, it is found that lesions of the lower right quadrant region are probably the most common of all. They may be divided into: first, appendicitis; second, incompetent ileocecal valve; third, dilatation of the cecum with retention; fourth, adhesions and angulations; fifth, ulcerations due to tuberculosis; and sixth, ulcerations due to carcinoma.

In acute appendicitis it is generally impossible to fill the appendix so that it can be visualized, but upon palpation under the fluoroscope a tender point will be found away from the colon and over an area that corresponds to the appendix position. The fundus of the stomach will

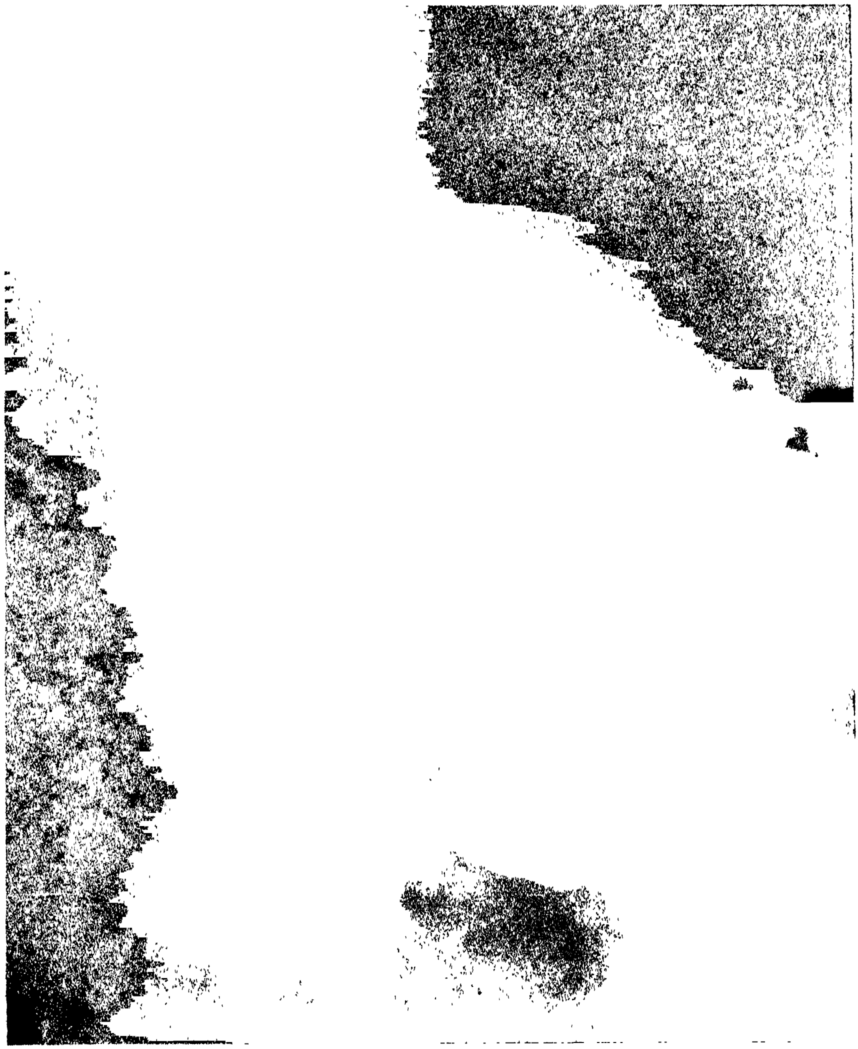

FIG. 31.—STOMACH PULLED IN THE LOWER RIGHT QUADRANT REGION. THIS STOMACH COULD BE PUSHED TO ITS NORMAL POSITION, INDICATING THERE WERE NO ADHESIONS, BUT WHEN RELEASED WOULD IMMEDIATELY RESUME ITS POSITION IN THE LOWER RIGHT QUADRANT REGION.

frequently be in the lower right quadrant region and while freely movable, yet will return to the lower right quadrant region. Frequently very valuable information may be obtained by the examination of chronic appendiceal affections. This, of course, applies only to those cases in which the lumen is patent, while in some of the very chronic forms the lumen may be completely closed as a result of an obliterative process, and consequently the barium will fail to enter and the appendix naturally cannot be visualized.

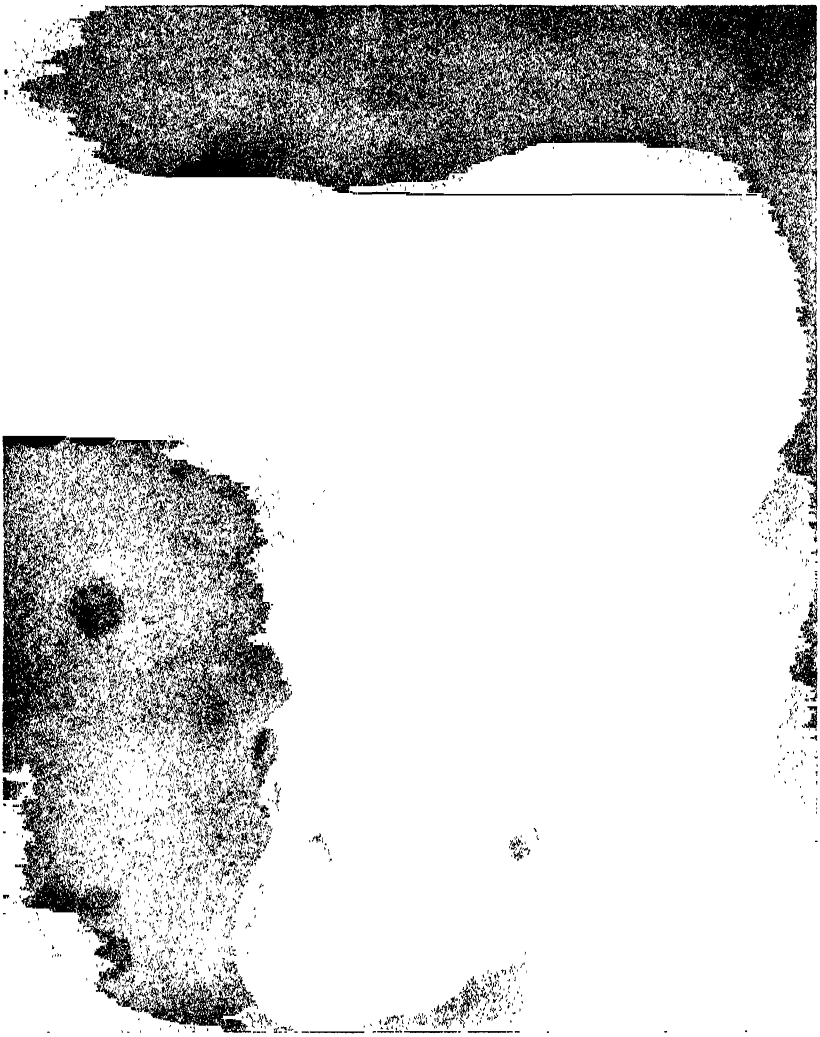

FIG. 32.—LARGE DILATATION OF THE CECUM WITH RETENTION OF CONTENTS.

In six hours after the administration of the barium meal the cecum is fairly filled, and at that time, under palpation, the appendix will probably fill. From that time on until the colon is empty and frequently for some time afterwards, the appendix remains visible. The ordinary technic frequently will not show the appendix, so that it must first be found fluoroscopically and then a plate made in the position in which it is visible. When the appendix remains visible for more than a day or two after the barium meal, it is in proportion to its poor drainage a dangerous appendix. There are certain instances in which the barium

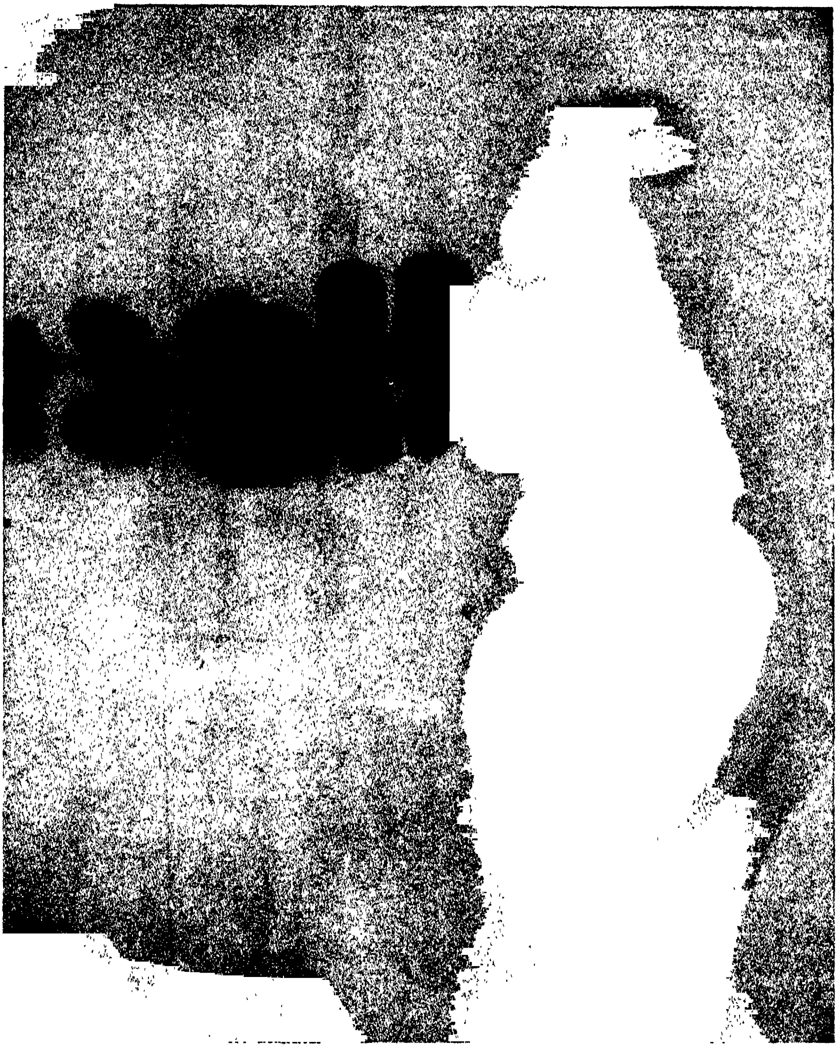

Fig. 33.--Adhesions Around the Ileocecal Valve Causing Fifteen Hour Retention and Some Dilatation of the

shadow may persist for days and in one or two instances the barium-filled appendix persisted for several weeks.

When visualizing the appendix the tenderness can often be localized immediately over it and, if the appendix be moved by palpation, the tenderness will move with it. When the appendix is retrocecal, the cecum must be pushed aside to visualize it (Fig. 29). The great majority of the retrocecal type show tenderness. The frequency of the visualization of the appendix by means of the barium meal varies according to several authorities from 35 to 90 per cent.

The author does not believe that every visualized appendix is necessarily pathological. An appendix can frequently be found well filled with barium, yet lying perfectly free, no signs of adhesions and emptying at the same time with the cecum. On the other hand, when we find an appendix filled with barium, curled up and fixed (Fig. 30), it invariably means that some pathological process is present. When chronically inflamed, an appendix is frequently adherent to the structures surrounding it. It may be fixed throughout its length or only partially so. It is a frequent occurrence to find the tip adherent with the body lying free. Palpation of such an appendix will almost invariably show tenderness.

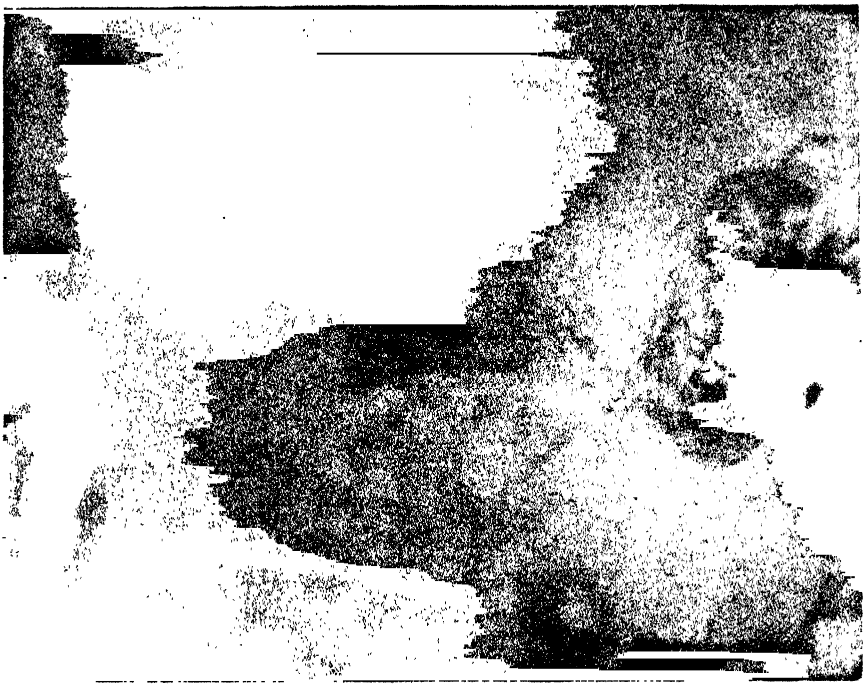

FIG. 34.—TUBERCULOUS ULCERATION OF THE LARGE BOWEL WITH HYPERMOTILITY. THIS PLATE WAS MADE SIX HOURS AFTER THE ADMINISTRATION OF THE BARIUM MEAL.

Kinking of the appendix is a significant sign of adhesions. When it points upward to the gall-bladder region, there is good ground to believe that some pathological condition is present.

Attention has been called to the fact that the fundus of the stomach is seen in the lower right quadrant region in acute appendicitis. This frequently is true of the chronic type. Occasionally this is due to adhesions to the stomach or omentum, and under palpation we will find it fixed in that position so that it cannot be moved. In other cases, when viewed under the fluoroscope screen, the stomach can be moved to its

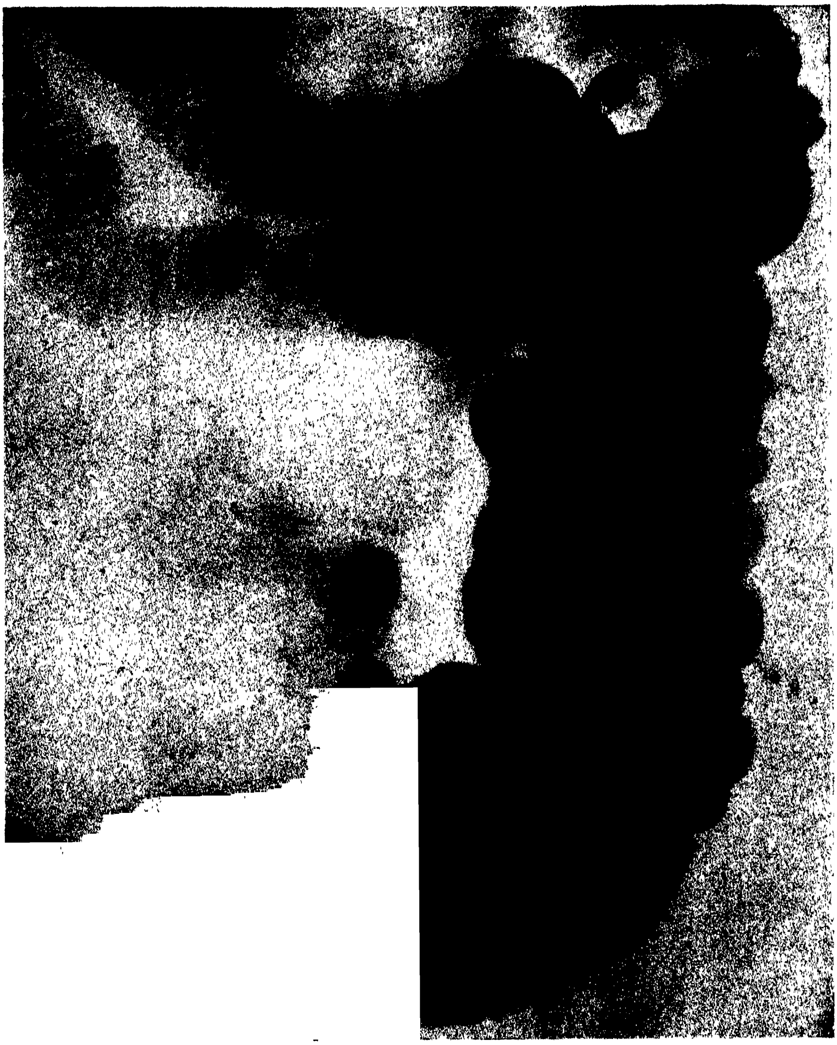

FIG. 35.—EARLY CARCINOMA OF THE CECUM INVOLVING THE ILEOCECAL VALVE.

normal position and yet upon the release of the hand will immediately move again to its former position (Fig. 31). The reason for this abnormal position cannot be explained, but it is probably due to some chemotactic attraction, and when present is a very definite indication of a diseased appendix.

Partial obstructions of the pylorus due to inflammatory conditions may lead to the production of adhesions in the lower right quadrant region, dragging the stomach in that direction, and the appendix may become secondarily affected. Adhesions associated with the appendix may extend to the surrounding structures and lead to varying degrees of cecal and ileal stasis, and even to partial colonic obstruction.

There are a certain number of cases of chronic infections where the appendix does not fill. This may be due to the fact that on account of its unusual position it does not fill, or its lumen is closed by an obliterative process, or for some other reason the barium does not enter. There may be even an absence of tenderness between attacks. Numerous authors have called attention to the fact that a diseased appendix may be present and yet not give roentgen signs. While tenderness over the appendix region is an important sign, it is well to remember that a chronically distended cecum is also tender upon palpation.

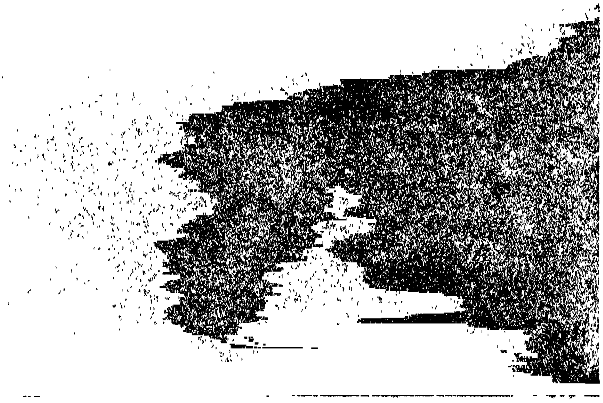

Fig. 36.—Carcinoma of the Transverse Colon Just Proximal to the Splenic Flexure.

Incompetent Ileocecal Valve.—When the barium has left the stomach and small intestines and is lodged in the colon and later examination shows the terminal ileum to be filled, we are dealing with a true ileocecal incompetency. When a barium enema is given it is quite common to find a part of the ileum filled. This must not be looked upon as a true incompetency, as the pressure of the enema plus antiperistaltic waves will produce this condition. The author feels that incompetency is of significance only when it occurs with the barium meal.

Dilatation of the Cecum with Retention.—When the barium meal is taken it ordinarily reaches the cecum in from six to nine hours. Delay

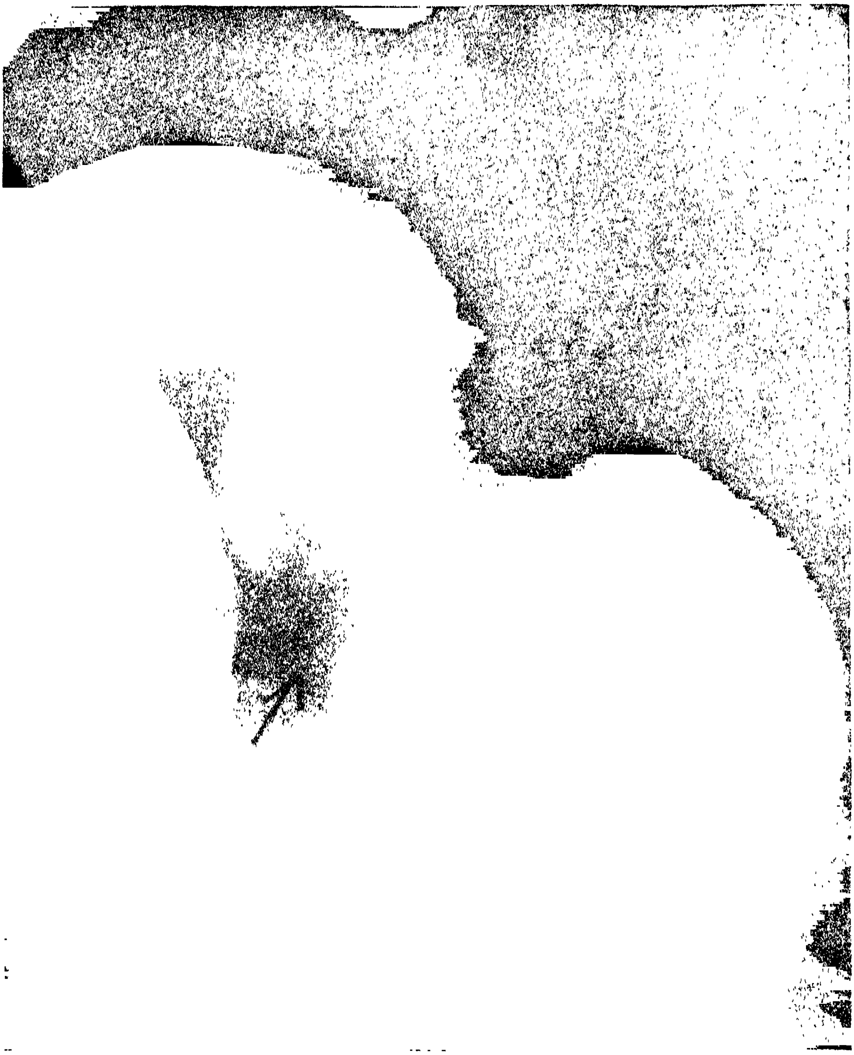

FIG. 37.—AN EXTENSIVE CARCINOMA OF THE SIGMOID PRODUCING PARTIAL OBSTRUCTION.

in the passage of the barium may cause a retention for many hours with consequent dilatation of the cecum (Fig. 32). This stasis may in some instances be associated with a chronic appendix and as T. R. Brown has pointed out both may be due to a high degree of enteroptosis, or they may be the result of a secondary low-grade inflammatory process. The degree of cecal stasis may vary markedly in various individuals, twenty-four or even forty-eight hours' retention not being unusual in some cases.

While the location of the cecum and colon is clearly pointed out, yet ptosis is a question of minor importance. It is not the position of the colon with which we are most concerned, but the functioning of this portion of the tract, for directly in proportion to disturbances of function are symptoms of retention and partial obstruction produced. The examination of the dilated cecum has probably thrown more light upon this type of constipation than has been done by any other means heretofore. It is interesting to note that with a dilated cecum the patient may frequently not complain of constipation, but on the contrary, state that the bowels move every day. Yet in these cases, one will find that a large part of the barium still remains in the cecum from forty-eight to one hundred twenty hours. The barium will become almost adherent in masses to the sides of the cecum, allowing only a small channel in the center. In some of the most obstinate cases when the cecum has been explored these fecal masses are so adherent to the walls that when removed there is sometimes bleeding of the mucous membrane beneath. This must of necessity cause a low-grade inflammation of the cecum, which will frequently extend to the appendix. In such cases on operation, when a chronically inflamed appendix is found, one must remember that the primary cause is not in the appendix, but in the cecum, and the removal of the appendix will not be of benefit to the patient.

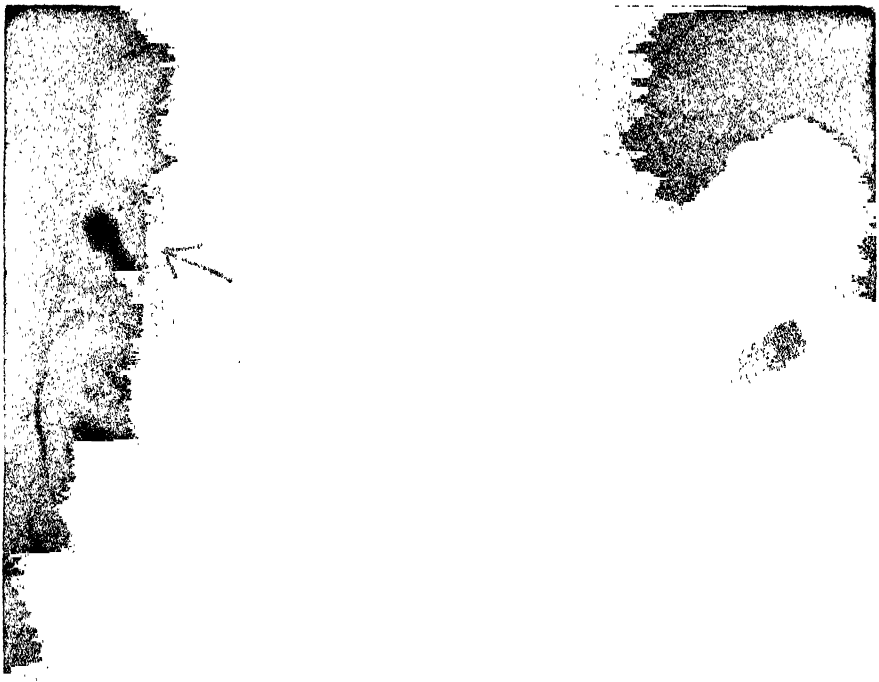

FIG. 38.—CHRONIC COLITIS, THE DESCENDING COLON BEING MORE OR LESS SUPERFICIALLY ULCERATED.

Adhesions and angulations can be readily recognized. Under the fluoroscopic screen the various parts of the bowels can be seen and when adherent they are fixed and cannot be separated. The adhesions may be so dense, particularly after an appendix abscess, as to produce partial obstruction, or the bowel may be pulled into such an abnormal position as to mechanically produce a partial obstruction. In adhesions from a chronic appendix, not only may the cecum be involved, but it is quite common to find the terminal ileum so bound down that there may be fifteen-hour retention and actual dilatation (Fig. 33).

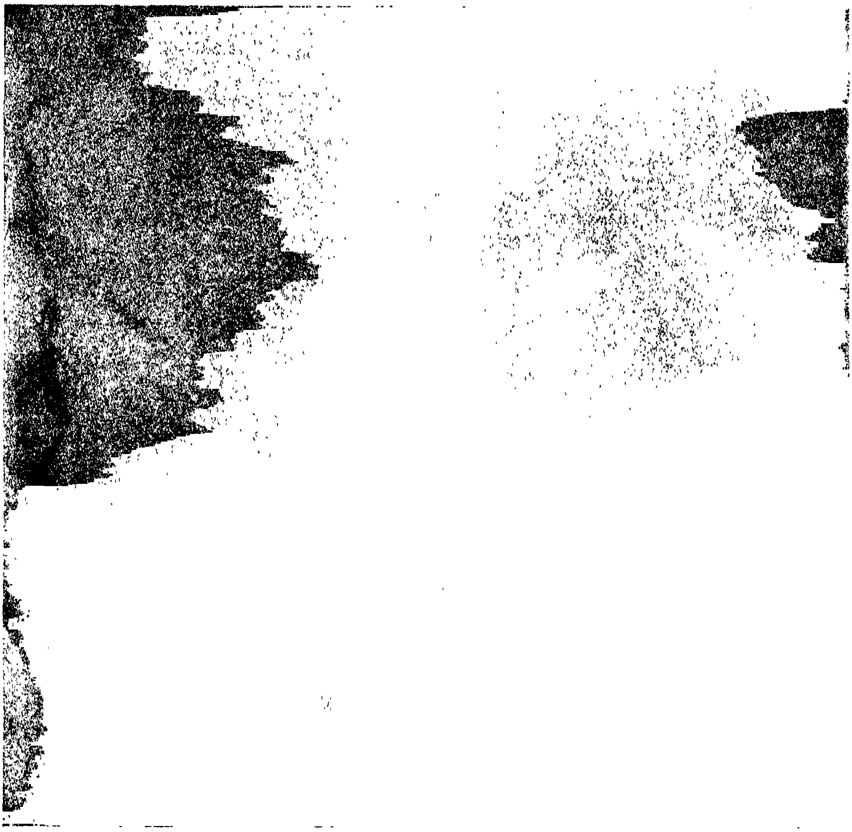

FIG. 39.—MARKED MUCOUS COLITIS, THE BARIUM BEING INCORPORATED IN MUCOUS SHREDS, GIVING A STRING-LIKE APPEARANCE TO THE TRANSVERSE AND DESCENDING COLON.

Tuberculous Ulcerations.—L. Brown and Sampson call attention to the importance of roentgen examination in the diagnosis of intestinal tuberculosis. According to these authors the clinical picture is of little aid in the diagnosis of the early stage of this disease. The roentgen examination shows hypermotility of the bowel, complete evacuation often

FIG. 40.—DIVERTICULITIS SHOWING GREAT NUMBERS OF DIVERTICULA, EXTENDING FROM THE WALLS OF THE SIGMOID AND THE TRANSVERSE COLON AND SPLENIC FLEXURE.

occurring in from twenty to twenty-four hours, though a small amount of barium may be left in the rectum at the end of this time. In many cases the cecum and ascending colon manifested the greatest hypermotility. The cecum and colon are very spastic and there are irregular filling defects due to ulcerative lesions. According to Brown and Sampson the presence of intestinal hypermotility spasm and filling defects gives almost definite evidence of colonic tuberculosis in a patient with pulmonary tuberculosis (Fig. 34).

Sometimes a very spastic, irritable bowel will frequently be improperly filled, due to spasm, and give a picture almost identical to that seen in tuberculosis, so that one must be careful not to assume that every case of spasm and improper filling is due to tuberculosis.

Ulcerations Due to Carcinoma.—While the barium meal gives us information as to the cecum, ascending and transverse colon, yet on account of the nonfilling of the descending colon and sigmoid it is of but little use in this area. The barium enema should always be used when a lesion is suspected in any portion of the colon. This method enables us to fill the colon completely and thus do away with areas of incomplete filling which may simulate the defects produced by carcinoma. While carcinoma may occur anywhere in the colon, the cecum (Fig. 35), transverse colon (Fig. 36), and sigmoid (Fig. 37) seem to be the favorite sites for this lesion, which is characterized by a filling defect with irregular serrated edges, the size depending upon the extent of the lesion. Carcinoma of the cecum may exist for a long period of time before obstructive symptoms are noted. In one or two instances the lesion was found upon routine examination where there were no clinical symptoms at all. Filling defects may occasionally occur from conditions other than carcinoma. In one instance the author saw a typical filling defect which was constant in all the examinations. The clinical history pointed to a lesion in the lower right quadrant. The filling defect and partial obstruction were found to be due to the dragging down of the colon by adhesion from an old inguinal hernia.

Diagnosis of carcinoma should never be made from a single examination. If the colon is not completely cleared the barium may flow around a small mass of fecal material which will give a filling defect. In such instances it is always well to wait several days and then have the colon thoroughly cleansed by means of cathartics and enemata, and repeat the examination. If the filling defect was due to a small mass of fecal material, it would have disappeared under brisk catharsis by the time the second examination was made.

We have spoken of adhesions in the lower right quadrant region, but they may occur anywhere in the abdomen. In gall-bladder infections we may see the hepatic flexure as well as the duodenum involved. Pus tubes may give marked adhesions in either lower quadrant according to the side involved. Occasionally fibrous bands will partially surround the colon and cause a partial stricture. They are recognized by the very abrupt narrowing of the lumen at the point of stricture, accompanied by a more or less constant spasm of the colon distal to the stricture.

Chronic colitis is best studied by the barium enema. In marked cases the colon is narrowed and has a "sausage-shaped tube" appearance. The usual haustration of the colon has disappeared due to the thickening of the wall from the chronic inflammatory process (Fig. 38). When the cecum is involved, there is generally incompetency of the ileocecal valve. In the milder forms of colitis the picture is just the reverse of the chronic type. There is marked spasm associated with deep haustration, and occasionally antiperistaltic waves. The barium becomes imbedded in shreds of mucus, giving a string-like appearance in the bowel (Fig. 39).

Diverticulitis is rather a rare condition and while any portion of the colon may be involved, the sigmoid is most frequently affected. The condition is characterized by small pouches opening into the colon (Fig. 40). These become filled with the barium and do not empty quickly. The barium in the colon will be expelled but there are left little round masses of barium along the edge of the colon. These may persist for a number of days before the pouches will empty their contents. In the advanced cases where there is marked induration we may get irregular filling defects which cannot be differentiated from those of cancer.

CHAPTER VII

DIET IN DIGESTIVE DISEASES

By Julius Friedenwald, M.D.

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DIET IN DIGESTIVE AFFECTION

General Consideration.—Diet plays an extremely important rôle in the treatment of digestive affections. In many of these disturbances cures can often be affected by diet alone, when without this mode of treatment serious complications may arise.

In order to more fully comprehend the value of diet in digestive diseases, it may be advisable to present briefly a few of the principles governing the use of food in general.

Foods are divided into a number of different classes, proteins, carbohydrates, fats, mineral salts and water. About 60 per cent. of the foods consist of water, which tends to maintain the proper consistency of the body; it does not, however, furnish heat or energy, as it is not burnt up in the metabolic processes. About 6 per cent. of the body weight of the adult is composed of mineral salts, the greatest amount being present as lime salts in the bones and teeth. The salts enter also, however, into the composition of other tissues and fluids of the body. While salts furnish but little energy and heat, they play an important rôle in metabolism, and are necessary for life and health, many diseased conditions being due to disturbances of salt metabolism. In addition to calcium other important salts are compounds of potassium, sodium, magnesia and iron.

Proteins.—Proteins comprise the food containing nitrogen. They are essential to life, and are split up during digestion into amino-acids, which are absorbed, and are utilized in the growth and repair of various tissues. A certain quantity of protein is requisite, but nitrogen equilibrium may be obtained at different levels, and if but little protein is ingested a part may be replaced by fats or carbohydrates, which are termed protein sparers.

Fats.—Fats are utilized to build up the fatty tissues. They are oxidized into carbon dioxid and water. Some of the fats may form sugar as in diabetes; others may be absorbed, and be stored away in the body. Part of the fats of the body is derived from the transformation of carbohydrates, while part is formed from protein.

Carbohydrates.—Carbohydrates are composed of carbon, hydrogen and oxygen, the last two in the proportion to form water. These foods include starches, sugar and vegetable fiber (cellulose). The carbohydrates may be transformed into fat and stored in the body, and may also be utilized as fuel.

Vitamins.—It is now known that the supplying of the body with adequate quantities of protein fats and carbohydrates is not sufficient to maintain growth and life. There are in addition, as Funk has pointed out, other substances which are essential; these are known as vitamins or accessory food substances. Three definite forms of vitamins have now been established:

(1) *Fat soluble "A"*, found in large amounts in milk, cream, butter fats, yolks of eggs, leaves of green vegetables, beef fat, and mutton fat.

(2) *Water soluble "B"*, or antineuritic, is observed in large quantities in eggs, many cereals, peas, sweetbreads and fish.

(3) *Antiscorbutic vitamin* is found especially in raw cabbage, lemon juice, orange juice, raspberries and tomatoes.

Fuel Value of Food.—Food has a definite fuel value as it is utilized or burnt up in the body. The calorie is the amount of heat that is necessary to raise the temperature of one kilogram (2.71 lbs.) of water 1° C., or nearly the same as the amount required to raise 1 pound of water 4° F. According to Rubner the caloric value of the primary food constituents is as follows:

1 gram protein	4.1
1 gram carbohydrate	4.1
1 gram fat	9.3
I gram alcohol	7.0

The caloric needs of the body under varying conditions may be stated as follows:

	Calories per Kilo (2.71 lbs.)	*Calories per Day for Man 65 Kilos (154 lbs.)*
Patient at rest in bed	28	1800
Repose	32	2100
Light work	33	2300
Moderate work	40	2600
Hard work	48	3100

Normally the appetite is a fair indication of the caloric needs of the body. In disease, however, this is not always true, for, due to lack of appetite, insufficient nourishment is consumed. In these instances, it need only be borne in mind that a patient resting quietly in bed requires far fewer calories than when up and about. This plan may be therefore taken advantage of in the treatment of undernourished patients and is an underlying principle of the so-called rest cures.

DIET IN DISEASES OF THE STOMACH

In the selection of the diet in diseases of the stomach, the food must be regulated not only in regard to the particular disease with which the patient is affected, but the taste and peculiarities of the individual must be taken into account. It may become necessary to make frequent changes in diet, but these must be cautiously and gradually made in conformity with the patient's ability to digest food. While there is but little difficulty in determining the digestibility of food under normal conditions, this is far more difficult in the various gastric disorders as the motor or secretory or both of these functions may be disturbed.

In arranging a diet list for patients with gastric disturbances two essential factors must be borne in mind; first the food must be prescribed in a very digestible form in order to lessen the work of the stomach; and second, it must be of such a character as to increase the patient's nutrition.

The well-known Leube diet scale is so arranged as to present the various foods in the order of their digestibility. This scale forms the basis of the Leube ulcer cure, and is of service in other conditions as well.

Leube's Diet Scale.—Diet I.—If the digestion is very much reduced, the following articles of food are most easily digestible: bouillon, meat solution, milk, raw or soft-boiled eggs.

Diet II.—Somewhat less digestible than Diet I are the following articles of food: boiled calves' brain, boiled thymus, boiled chicken, and pigeon. The different forms of meat are enumerated in the order of their digestibility. Gruels, and in the evening milk mushes made with tapioca and white of egg, may also be placed in this list. The majority of patients can digest boiled calves' feet in addition to the various meat foods already enumerated.

Diet III.—If Diet II is well borne, Diet III may be given. This consists in adding cooked or raw beef to Diet I. Leube gives the following method of preparing beefsteak, and believes that beef cooked in this way is very easily digested: The meat should be kept for some time, and then scraped with a dull spoon; in this way a pulp is obtained, consisting only of the delicate parts of the muscle, and not containing any of the tough, hard and sinewy portion; this pulp is roasted in fresh butter. Raw ham is also to be recommended. In addition to meat a small quantity of mashed potatoes may be given, some stale wheat bread and small amounts of coffee or tea with milk (cautiously).

Diet IV.—This list is so arranged that if the patient can digest the articles of food mentioned under this head for some time, he can then begin with his usually accustomed diet: Roast chicken, roast pigeon, venison, partridge, roast beef (medium to raw, particularly cold), veal (from the leg), pickerel, boiled shad (trout even when young is very difficult to digest), macaroni and bouillon with rice. Small quantities of wine may be taken one to two hours before eating; gravies are contra-indicated. Young and finely chopped spinach is allowable; other vegetables, such as asparagus, may be tried cautiously, although Leube considers this a rather risky procedure. After this fourth diet the patients are allowed to take a more liberal diet, but the increase should be gradual. They should refrain from eating vegetables, salads, preserves and fruits for some time; and when these are resumed, a baked apple is the first of these articles to be eaten.

Penzoldt has devised the following table giving the digestibility of food. He experimented on normal cases, achieving his results by means of the stomach tube, by determining the progress of digestion and the exact time at which the stomach was entirely empty after eating a certain quantity of special food. The table shows the period of time it takes a given quantity of food to leave the stomach:

One to Two Hours:	
Pure water	100-200 grams
Carbonated water	220 grams
Tea alone	200 grams
Coffee, alone	200 grams
Beer	200 grams
Light wines	200 grams
Boiled milk	100-200 grams
Meat broth, alone	200 grams
Egg, soft	100 grams
Two to Three Hours:	
Coffee with cream	200 grams
Cocoa with milk	200 grams
Malaga wine	200 grams
"Ofner" wine	200 grams
Water	300-500 grams
Beer	300-500 grams
Boiled milk	300-500 grams
Eggs, raw and scrambled, hard boiled or as omelet	100 grams
Beef sausage, raw	100 grams
Calves' brains, boiled	250 grams
Calves' thymus, boiled	250 grams
Oysters, raw	72 grams
Carp, boiled	200 grams
Pike, boiled	200 grams
Shellfish, boiled	250 grams
Cod, boiled	200 grams
Cauliflower, boiled	150 grams

Two to Three Hours (Cont'd):

Cauliflower, as salad	150 grams
Asparagus, boiled	150 grams
Potatoes boiled in salt water	150 grams
Mashed potatoes	150 grams
Stewed cherries	150 grams
Raw cherries	150 grams
White bread, old or fresh, dry or with tea	70 grams
Pretzels	70 grams
Zwieback, fresh or stale, dry or with tea	70 grams
Albert biscuits	50 grams

Three to Four Hours:

Young chicken, boiled	230 grams
Partridge, boiled	230 grams
Pigeon, boiled	220-260 grams
Pigeon, fried	195 grams
Beef, raw, boiled, lean	250 grams
Calves' feet, boiled	250 grams
Ham, boiled	160 grams
Veal, warm and cold	160 grams
Veal, warm and cold, lean	100 grams
Beefsteak, broiled, cold or warm	100 grams
Beefsteak, raw, scraped	100 grams
Tenderloin	100 grams
Rhine salmon, boiled	200 grams
Caviare, salted	75 grams
Sardines in vinegar, kippered herring	200 grams
Black bread	150 grams
Barley bread	150 grams
Wheat bread	150 grams
Albert biscuits	100-150 grams
Potato as vegetable	150 grams
Rice, boiled	150 grams
Kohlrabi, boiled	150 grams
Carrots, boiled	150 grams
Spinach, boiled	150 grams
Cucumber salad	150 grams
Radishes, raw	150 grams
Apples	150 grams

Four to Five Hours:

Pigeon, broiled	210 grams
Fillet, or beef broiled	250 grams
Beefsteak, broiled	250 grams
Beef tongue, smoked	250 grams
Smoked beef in slices	100 grams
Hare, broiled	250 grams
Partridge, broiled	250 grams
Goose, broiled	250 grams
Duck, broiled	280 grams
Herring, salted	250 grams
Lentils, mashed	150 grams
Peas or purée	200 grams
String beans	150 grams

In prescribing a diet for patients affected with gastric disturbances it is important that the food be rapidly dissolved in the gastric secretion, that it be passed into the intestine at a normal rate, and be quickly absorbed. This process must be accomplished without abnormal fermentation or discomfort. The digestibility of any particular food depends largely upon the individual taste, for no matter how digestible a food may be, it cannot be perfectly digested if it is unpalatable to the patient.

It is important to conform the diet according to the special variations in the functions of the stomach. The effect of the food upon the motor and secretory functions depends to a great degree upon its structure and chemical composition; the more finely comminuted the food the less it is apt to overtax these functions.

In prescribing the diet in gastric affections attention must be called to the fact that in acute conditions the food should be so constituted that the stomach be spared as far as possible, while in chronic disturbances it is important to supply in an easily digestible form a sufficient quantity of nourishment to maintain the body weight. Inasmuch as at rest an individual requires 35 calories per kilo of the body weight and in performing light work 40, in estimating the quantity of nourishment needed, it is necessary to know only the weight of the individual. In so far as the protein can be replaced to a certain degree by the carbohydrates and fats, an interchange of any of these foodstuffs may be made, which should vary in accordance with the condition of the patient.

König's tables giving the composition of various foodstuffs and their caloric value are interesting in this connection. According to the author's experience in keeping a record of the weight of the patient a clear idea can be obtained as to whether a sufficient amount of nourishment is taken. It has been his habit to weigh every patient affected with digestive disturbances as soon as treatment is instituted and from time to time again to determine whether the patient is gaining or losing flesh.

Composition of the Most Common Food Substances

Dairy Products	Protein (*Per cent.*)	Fat (*Per cent.*)	Carbohydrates (*Per cent.*)	Calories (*Per cent.*)
Cow's milk	4-4.4	3.00-3.80	3.70	64.00
Cream	3.61	26.75	3.50	276.01
Butter	0.50	90.00	0.50	837.00
Whey	0.50	0.30	3.60	
Buttermilk	3.00	1.30	3.00	3.67
Kumiss or cow's milk	3.35	2.07	0.70 Lactic acid 1.90 Alcohol 0.80 Carbonic acid	32.99
Cheese, cream	25.00	30.00	3.00	394.00
Cheese	33.00	9.00	5.00	240.00
Eggs	12.50	12.00	0.50	165.00
Meat and Game				
Beef (fat)	17.19	26.38	....	315.81
Beef (lean)	20.78	1.50	0.07	99.15
Veal (fat)	18.18	7.41	....	146.61
Veal (lean)	19.84	0.80	....	86.97
Mutton (very fat)	14.80	36.39	0.05	399.31
Mutton (lean)	17.11	5.79	....	120.81
Pork (fat)	14.54	37.34	....	406.88
Pork (lean)	20.25	6.81	....	146.30
Ham (Westphalian)	23.97	36.48	1.50	453.69
Sweetbread	22.00	0.40	....	93.92
Pulverized meat	64.50	5.24	2.28	322.53
Poultry	22.00	1.00	....	100.00
Spring chicken	18.49	9.34	1.20	167.59
Duck	22.65	3.11	2.33	131.36
Squab	22.14	1.00	0.76	100.07
Game	23.00	1.00	....	103.60
Fish				
Pike	18.50	0.50	0.75	83.57
Carp	20.61	1.09	....	94.64
Shellfish	17.09	9.34	....	
Salmon	15.01	6.42	2.85	156.93
Sardellen	22.30	2.21	0.45	132.93
Oysters	4.95	0.37	....	
Salt herring	19.50	17.00	0.50	24.00
Caviare	28.04	16.26	7.82	

COMPOSITION OF MOST COMMON FOOD SUBSTANCES (CONT'D)

Cereals and Vegetables	Proteins (*Per cent.*)	Fat (*Per cent.*)	Carbohydrates (*Per cent.*)	Calories (*Per cent.*)
Sago	0.50	Trace	86.50	356.70
Wheat flour	8.50	1.25	73.00	354.78
Rye flour	10.00	2.00	69.00	342.50
Wheaten bread R	6.00	0.75	52.00	245.00
Rye bread	4.50	1.00	46.00	216.00
Zwieback	9.50	1.00	75.00	356.00
Roll	6.82	0.77	43.72	213.87
Cauliflower	200-500	0.40	4.00	35.00
Carrots	1.04	0.21	6.74	33.85
Asparagus	2.00	0.30	2.50	21.00
Rice	5.50	1.50	76.00	348.10
Beans	19.50	2.00	2.00	311.75
Peas	19.50	2.00	54.00	319.95
Potatoes	1.50	....	20.00	88.00
Oatmeal	12.50	5.26	66.77	338.80
Barley meal	8.31	0.81	74.19	323.00
Spinach	3.49	0.58	4.44	38.00
Pickles	1.02	0.09	0.95	
Soups and Beverages				
Milk soup with wheat flour	5.00	3.25	15.00	112.00
Meat broth (ordinary)	0.40	0.60		
Meat juice (pressed)	6.00-7.00	0.50		
Beef tea	9.00-11.00			
	Protein			
Leube's meat solution	1.79-6.50			
	Peptone			
	8.00-10.00			
Barley soup	1.50	1.00	11.00	60.96
Malt extract	....	....	55.00	258.30
Rice pap, with milk	8.80	3.50	28.00	182.61
	3.12	5.18		
	12.38			
	0.50	5.25	0.30	
	0.70	6.00	0.30	6.00
Fruits	Free Acid (*Per cent.*)	Protein (*Per cent.*)	Fat (*Per cent.*)	Carbohydrates (*Per cent.*)
Apples	0.82	0.36	....	7.22
Pears	0.20	0.36	....	3.54
Plums	1.50	0.40	....	4.68
Peaches	0.92	0.65	....	7.17
Grapes	0.79	0.59	....	1.96
Strawberries	0.93	0.54	0.45	1.01
Chestnuts	....	5.48	1.37	38.34
Sugar cane	....	....	....	3.40
Honey	....	1.20	....	5.28

The character of diet prescribed must be considered from the standpoint of gastric secretion; that is, whether we are dealing with a condition of hyperacidity on the one hand or subacidity on the other. In hyperacidity an abundant protein diet is advisable in order to neutralize the excess of hydrochloric acid. This is not always the case, however, for we have observed that these patients at times do better on bread and amylaceous food. The proteins which are of the greatest value in cases of hyperacidity are eggs and red meats, while the carbohydrates must be allowed only in small amounts in a most digestible form. In cases of subacidity the proteins as a rule are most difficult to digest while the carbohydrates are more easily digested. In these cases the meat should be allowed in such tender forms as scraped beef, boiled sweetbreads, brains, etc., while the vegetables are permitted in larger quantities. In both conditions of hyperacidity and subacidity a certain amount of good butter is allowable. This form of food is especially valuable in cases of hyperacidity as it has a marked tendency to lessen the quantity of hydrochloric acid secreted.

In motor disturbances of the stomach the diet to be prescribed depends greatly as to whether there be at hand an excess or diminution of acid in the gastric secretion. If there is an excess of acid an increase of protein food is advisable. If there is a diminution of this secretion the proteins must be given in the most digestible form, while the carbohydrates and lighter vegetables may be allowed in large quantities. The author agrees with most authorities that ingestion of fluids in all motor disturbances of the stomach should be reduced as far as possible. They should be taken in small quantities and largely between meals. While normally the appetite is usually a fair indication of the number of heat calories required, this cannot be said to be true whenever gastric disorders exist, for due to the impairment of the appetite insufficient nourishment is often consumed. The fact that a patient at rest in bed requires fewer calories than is necessary when he is at work can be taken advantage of in the treatment of protracted gastric disturbances in which a state of subnutrition exists. Very satisfactory results are obtained in many of these cases by placing such a patient at rest in bed for a certain length of time.

Liquid Food in Gastric Disturbances.—Milk is the food which is usually best borne whenever it is necessary to prescribe liquids in order to spare the stomach. When given alone, large quantities must be consumed in order to supply the requisite amount of nourishment. It is best taken diluted with lime water or Vichy, or citrate of soda may be added in order to render it more digestible, or it may be flavored with tea, coffee or vanilla to add to its palatability. In those instances in which milk is badly borne, buttermilk, kefir or kumiss may be used as substitutes. Of the other liquid foods which may be utilized are the various forms of broths, meat juice or beef tea; of these meat juice is decidedly more nutritious. Tea and coffee should only be taken weak and may serve as a means of flavoring milk. Cocoa is highly nutritious and serves as a valuable liquid food. The various forms of meat may be made more digestible by scraping and grinding.

Gelatinous Forms of Food.—Gelatin, calves' foot jelly and chicken jelly are more easily digestible. Eggs either raw or soft boiled are most digestible, though they are at times preferred in the poached or scrambled forms. Fish, which contain but small amounts of fat, are to be recommended, as halibut, shell fish, and trout.

In order to render vegetables digestible, they must be thoroughly masticated; they should be given cautiously, whenever there is marked fermentation, perhaps best in the purée form. Inasmuch as leguminous foods are apt to give rise to fermentation, care must be exercised regarding their use. Cabbage should be prohibited from the dietary of patients affected with gastric affections. Potatoes are most easily digested in baked or mashed form. Amylaceous food is best prescribed as toast, oatmeal, tapioca, etc. Fruits, while they give relish for other food and increase the bowel movements, have but little nutritive value; yet they are rich in vitamins, especially of the antiscorbutic type and are therefore important as accessory food substances.

When fat is taken in certain forms it produces indigestion; this condition especially occurs after the use of fat meats, greasy gravies, etc. Such food should be omitted from the dietary of patients affected with gastric disorders. In the proper form fat is utilized to advantage in increasing the body weight and at the same time it tends to decrease the acidity of the stomach. Fats can be utilized in the form of good butter, taken on bread, and chocolate and olive oil.

SPECIAL FACTORS RELATING TO THE DIET OF PATIENTS AFFECTED WITH GASTRIC DISTURBANCES

When the gastric secretion is lost, the intestines will vicariously perform the work of the stomach, so that a patient with this form of disturbance may maintain his weight. This fact may easily be demonstrated in cases of achylia gastrica. When it is necessary to spare the stomach as when food is vomited or not digested, nourishment may be administered by channels other than the stomach as by rectal feeding, or it may be administered directly into the duodenum through the duodenal tube.

It is important that the food should be thoroughly masticated not only to divide it properly, but also to increase salivary secretion and digestion which in turn have an important influence on the gastric functions. The temperature of the food is also of primary importance, and it should be taken only at a temperature of between 98° and 100° F. (36.7° and 37.8° C.). As a rule the meals should be of moderate size, and taken at regular intervals.

The question of rest and exercise is of considerable importance to those suffering with gastric disturbances. From the author's observation the following conclusions are drawn:

(1) In persons with normal digestive powers it makes little difference whether the individual rests, exercises or sleeps after meals, though after violent exercise or sleep the gastric digestion is very slightly impaired.

(2) In patients suffering with superacidity and subacidity it is best to order rest after meals; after violent exercise or sleep the digestion is impaired in these cases.

(3) In patients suffering with motor disturbances of the stomach

it is best to prescribe moderate exercise after meals, for rest, violent exercise, or sleep disturb the digestion under these conditions.

SPECIAL FORMS OF TREATMENT IN GASTRIC AFFECTIONS

The most important forms of special cures utilized in the treatment of gastric affections are the **rest cure, milk cure, forced feeding, duodenal and rectal alimentation.**

Rest Cure.—The rest cure first devised by Weir Mitchell is a valuable form of treatment in gastric affections. The author is constantly utilizing it in the various forms of gastric neurasthenias accompanied by severe anorexia and emaciation with gratifying results. It is especially valuable in the treatment of ulcer, gastritis, atony and gastroptosis.

The technic of the treatment is explained in a most interesting way by Mitchell, in his book "Fat and Blood." The more nearly perfect the technic and the more closely it is adhered to, the more likely is a cure to follow. The cases are of various grades of severity, and the treatment is modified to suit the individual. Isolation is necessary, and the patient should be removed to a **hospital** or **sanitarium** away from familiar scenes.

Home treatment does not succeed well. If the circumstances compel the patient to remain at home, her **room should be changed.** In severe cases with emotional manifestations **visiting is forbidden,** but it may be allowed to a "certain extent when the patient is anemic owing to a distinct cause, as overwork, blood losses or dyspepsia." The **nurse** should be a **stranger** to the patient.

Communication with friends and family should, as a rule, be cut off entirely, and not even the reading of letters should be allowed. After several weeks, if the patient is improving, she may be allowed to read the newspaper each day. Rest is a most important feature, and, as a rule, the patient should **be put to bed for six or eight weeks.** The results following this form of treatment are usually most gratifying.

The diet should be varied, food being supplied at two- or three-hour intervals. At first it is well for the patient to be placed on a strict milk diet, supplying this food in large quantities. The caloric value of the milk may be greatly increased by the addition of cream. The diet should contain proteins and in addition be rich in fats and carbohydrates, and in most instances whenever the gastric condition permits, overfeeding must be enforced.

Milk Cure.—This cure consists in the administration of large quantities of milk, taken either alone or together with other food. In a number of gastric disorders milk may be taken alone, allowing the stomach to regain its normal functions. It is especially of value in the treatment of ulcer of the stomach, and some forms of chronic gastritis, and also in the secondary forms of gastritis, dependent upon tuberculosis and anemia. In certain forms of gastric neurasthenia the milk cure produces gratifying results, while in other conditions milk quickly disturbs the digestion, and cannot be employed.

It is especially badly borne in those affections in which there is a decrease or absence of acid in the stomach. The milk cure is contraindicated in severe forms of atony and in dilatation as well as in intestinal affections associated with marked flatulency and in chronic diarrhea. When milk is taken in large quantities in addition to other food, it is much less apt to disagree, and may produce constipation which can be overcome by the addition of milk of magnesia. When it is not well digested, lime water, citrate of soda or barley water may be added, and the milk may be flavored with tea, coffee or cocoa, or buttermilk may be substituted for it.

Forced Feeding or Lavage.—This method consists in **introducing milk, eggs, broth into the stomach through the stomach tube.** On occasions the author has employed it in aggravated cases of nervous anorexia and sitophobia where there seemed to be danger of starvation.

Duodenal Feeding.—Of the greatest importance in the treatment of certain forms of gastric disorders is the method devised by **Einhorn,** known as **duodenal alimentation.** By means of this method food can be introduced directly into the duodenum. The instrument employed consists of a small capsule, perforated and attached to a long rubber tube, at the other end of which a syringe can be applied. The tube is swallowed along with water; the instrument soon passes into the stomach and within an hour or two into the duodenum. Care should be taken to see that it is in place before the feeding is started. This may be done as follows: by gentle traction, which shows a slight resistance if the tube is in the duodenum; by aspiration, which will often bring up golden-yellow duodenal juice without any gastric secretion; or, perhaps best by giving the patient some liquid to drink by mouth and immediately performing aspiration—if the end of the tube is in the stomach, the fluid can be removed. Any liquid food may be employed, but mixtures of milk-sugar and raw eggs are the most useful. Care should be taken to see that there are no particles in the food that might clog the tube. The amount at the start should be small, 100 c.c. (3.38 fluid ounces) every two hours. Feeding should be begun early in the morning and stopped late in the evening. This quantity may be gradually increased up to 300 c.c. If eight feedings are given in twenty-four hours and each feeding consists of 280 c.c. of milk, one egg, and one tablespoonful of sugar of milk, the patient will receive approximately 2280 calories, which is ample for an average individual. Furthermore, if the patient is at rest in bed, it is sufficient to allow a gain in weight.

Einhorn has perfected a special syringe with which it is possible to administer the food without disconnecting the tube. Morgan has suggested a method like that of Murphy for giving salt solution per rectum, permitting the fluid to flow from an irrigating jar and so arranging the petcock that the food is taken slowly, the 300 c.c. of nourishment being consumed in about twenty-five minutes. The food should be administered at body temperature, and the heating should be done slowly for if it becomes too hot it is liable to become thick and lumpy. After heating, it is well to strain the food to be certain to have it free from small particles. If the food is used too warm or too cold, it is apt to produce uncomfortable symptoms, sometimes causing the patient considerable shock; a too rapid administration causes flatulence. After each feeding a syringeful of water at 98° F. (36.7° C.) should be injected; then the petcock should be closed, and the syringe filled with air; this should be injected after the petcock has been opened; then the petcock should be closed, and the syringe disconnected. This procedure is very important and serves to keep the tube clean and empty.

Rectal Feeding.—When the patient cannot take food by the mouth or when it is not desirable to feed him in that way, rectal feeding is resorted to. All the various classes of foods may be used for this purpose. Proteins are supplied in the form of **predigested meat,** or **egg albumen** to which salt has been added. **Nutrose** and **peptones** are also used. Grape sugar and starch are sometimes employed. Fat is given in the form of the yolk of an egg, or cream. Milk is also much used. Where the patient is to be fed by the rectum for any length of time, combinations of these are used as suggested in the formulas given below. Baüer believes that but one-fourth of the nutriment needed by the body can be absorbed by the rectum, and both he and the earlier writers placed the length of time during which rectal feeding was practicable at from one to two weeks. With careful technic, this period may be extended from four to six weeks, depending upon the capacity of the individual for continued absorption, and on the amount of energy stored up in his body at the beginning of the rectal feeding; but von Leube has kept a patient alive for six months, and Riegel for ten months on exclusive rectal feeding. As a rule, however, as only about one-fourth, and often even less, of the amount of nutriment needed can be absorbed, the method is useful only in protecting the body from excessive loss during periods of partial or complete starvation. The question of the absorption of the various foods from the rectum has not been definitely settled, and is deserving of further study, but it would seem that the **carbohydrates** are absorbed better than any of the others, something like nine-tenths of the total quantity injected being absorbed when 10 to 20 per cent. sugar solution is used; but not more than 40 grams of sugar can be given by rectum, and as a rule, a daily allowance of 30 grams is about as much as the average patient can tolerate. Solutions of pure **glucose, dextrose,** or **dextrin** have given the best results, and **boiled starch** is also frequently used in conjunction with other things. The proteins are not absorbed very well, only about one-sixth of that given being utilized, although certain forms of dried casein seem to be absorbed to quite a remarkable degree. Fats are absorbed with difficulty, but doubtless better than was formerly supposed. They are particularly useful on account of their high food value. Various fats and oils have been suggested, both emulsified and saponified, but the **yolk of eggs** seems to give the best results. **Alcohol** is usually easily absorbed in ½ to 2 per cent. solutions, and in many instances apparently aids in causing the absorption of other foods. An important addition to all nutrient enemas is **ordinary salt** which should be added up to 1 per cent. This aids materially in the absorption, and also apparently favors reverse peristalsis, which causes the food to be carried upward in the bowel, and so brings it in contact with a greater surface of the intestine. The temperature of the solution injected is very important. If the solutions are either too hot or too cold, they will be quickly rejected. The solutions intended for retention should be between 90° and 95° F. (32.2° and 35° C.).

The **technic of rectal feeding** is exceedingly important, and to be carried on successfully over any period of time must be done by some one having experience. The enema should be preceded by a cleansing injection, which ordinarily may be of the strength of 1 dram (3.9 grams) to the pint. Sodium bicarbonate may be used if there is much mucus in the rectum, and sometimes boric acid solutions may be employed to advantage. The first two or three cleansing injections should be given with an ordinary rectal tube; after that they may be given to great advantage through a return flow tube. The temperature of these cleansing injections should be between 95° and 99° F. (35° and 37.2° C.). The patient should be placed in as comfortable a position as possible, a somewhat modified knee-chest position being used, with a large pillow so placed as to elevate the hips, and a small rectal tube lubricated with oil introduced slowly as high into the rectum as possible. Glycerin should never be used as a lubricant. The tube should be passed well beyond the inner sphincter. It is not necessary to push in great lengths of tubing, as it only curls up in the rectum. Only in exceptional cases does it pass into the sigmoid, and certainly not unless fluid is allowed to flow through the tube at the time it is introduced; and this cannot be done when the tube is passed for purposes of rectal feeding. The solution used should be allowed to flow in slowly. One should use a fountain syringe connected with the rectal tube, and only slightly elevated above the hips of the patient, who should remain as quiet as possible after the injection, and if possible, should be diverted by conversation or other means. If necessary, a pad of gauze or a towel may be held over the anus for twenty or thirty minutes where the injections are poorly retained. Where there is irritability of the rectum, or when the injections cause pain, they must be preceded by a suppository containing **opium**, or a **small enema of boiled starch**, to which opium has been added, may be used. Where there are hemorrhoids which are painful, the mucous membrane may be painted with a 2 per cent. solution of **cocain**. It should be borne in mind that gynecological dressings may interfere with rectal feeding.

The number of nutrient enemata which may be used depends somewhat upon the individual patient. Ordinarily, three or four may be given in the course of twenty-four hours, at about six-hour intervals. Where the patient is badly in need of food, the interval may be made five hours, and it is only in exceptional instances that they should be given closer together than this, as too frequent use of injections leads to irritability of the rectum. The amount to be given at any one time should be 8 ounces (248.8 grams), or less.

A part of the good effect of the nutrient enemata is the mental satisfaction following it. It is, in fact, similar to that following a meal, and the patient also has the feeling that he is not being allowed to starve.

Nutrient Enemata by the Drop Method.—Since Murphy's introduction of continuous proctoclysis by the drop method, the administration of nutrient enemata by the same plan has been practiced by various clinicians. Eberhard has called attention to this method of treatment. His apparatus consists of an ordinary quart can, inside of which is placed another can holding a pint. These are connected by an 8 inch (20.08 cm.) pipe, which penetrates the bottom of each and projects about 2 inches (5.09 cm.). A small petcock, soldered to the base of the outside can, allows water to be withdrawn at will. Milk and egg or any other nutrient placed in the smaller can is kept warm, and flows freely on account of its being surrounded by hot water. Water at a temperature of from 110° to 115° F. (43.3° to 46.1° C.) seems to answer all purposes. The remainder of the apparatus is the same as is used for saline enteroclysis. To insure absorption the bowel must be cleansed by an enema each day. The flow must be regulated to about a drop a second. According to Eberhard, in one and a half hours 10 ounces (331 grams) of milk and 2 raw eggs flow into the bowel. Eberhard has had the best results from the following enemata:

Albumen of 3 eggs	90 calories
Peptonized milk, 9 ounces (280 grams)	174 calories
Table salt, ½ dram (1.95 gram)	9 calories
	273 calories

The following formulæ for preparing nutrient enemata have been found useful:

Formulæ after Smithies

1. Milk pancreatized for twenty-four hours and boiled	250 c.c.
2. Add to the above either alone or in combination:	
(*a*) Dextrose	5 to 12.5 grams.
(*b*) Alcohol	5 to 12.5 grams.
(*c*) Egg yolk	1 to 2 c.c.
3. Alcohol (50 per cent.)	1 ounce
Glucose syrup	1 ounce
Normal salt solution	6 ounces

DIET IN ACUTE GASTRITIS

In the treatment of this affection diet plays a most important rôle. The indications are to secure an absolute rest for the stomach as well as total abstinence from food for at least twenty-four hours. This is usually a difficult procedure, for most patients hold the belief that food is necessary for them, and are unwilling to abstain from nourishment for a sufficient length of time. It does not, however, take long to convince the patient of the need of abstinence from food inasmuch as nausea and vomiting are aggravated by the ingestion of nourishment.

For the thirst which is marked in this affection, the patient may be allowed to rinse his mouth frequently with water or even to take very small quantities of Vichy or Apollinaris. According to the author's experience the patient will usually recover in three or four days, when following this plan of treatment. After the first twenty-four or forty-eight hours, small quantities of milk diluted with lime water are permitted, or Vichy or citrate of soda may be added (a grain to the ounce of milk), or plain albumen, or clear broths allowed. These are gradually increased and following this scraped beef, chicken, sweetbreads and toast may be allowed. The following table taken from "Diet in Health and Disease," by Friedenwald and Ruhräh, gives the diet about the third and fourth day after an attack of acute gastritis:

Time	Grams	Food	Calories
7 A. M.	150	Milk with lime water	101
9 A. M.	100	Egg albumen flavored with orange or lemon juice	54
11 A. M.	150	Broth with egg	84
1 P. M.	150	Milk with lime water	101
3 P. M.	5	Armour's soluble beef in water	10
5 P. M.	100	Egg albumen flavored with orange or lemon juice	53
7 P. M.	150	Milk with lime water	101
			504

After the third day the diet is increased as follows:

Time	Grams	Food	Calories
7 A. M.	150	Milk (101) with 70 grams toast (182)	283
9 A. M.		2 very soft boiled eggs	160
11 A. M.	200	Bouillon with 1 egg	83
1 P. M.	100	Rice cooked in milk	177
	70	Toast	182
3 P. M.	100	Egg albumen (53) with 50 grams of cracker (187)	240
5 P. M.	150	Milk with 70 grams of toast	283
7 P. M.	100	Egg albumen flavored with orange or lemon juice	53
			1461

DIET IN CHRONIC GASTRITIS

In this affection the dietetic treatment is of far greater importance than the use of drugs. The most digestible forms of foods are liquids such as broth and their food value may be increased by the addition of compressed beef juice, barley, rice and eggs. The diet should be varied ordinarily according to the severity of the disease.

In those forms in which there is a true achylia, proteins are digested with great difficulty and should be allowed only in the most digestible form. Foods such as sweetbreads, steamed chicken, and scraped beef are especially valuable. Vegetables are best taken in the purée form. Usually milk is well borne and serves as a useful food.

When the gastric secretion still contains acid, meats form an important part of the diet, and any of the more easily digested meats may be allowed. Fish and eggs may be added to the dietary and such vegetables as spinach, mashed and baked potatoes, and carrots and peas, mashed and strained, are to be recommended. Cereals when well cooked are also useful foods in this affection. In order to supply the necessary amount of fat, butter, olive oil and chocolate are valuable.

It is best according to the author's experience to prescribe small meals given at frequent intervals. Mineral waters are useful in the treatment of certain forms of chronic gastritis, and of these the saline waters and alkaline saline waters are especially to be recommended. Saratoga (Congress and Hawthorn) and Bedford Springs water are among the important waters which the author observed as being of some benefit in the treatment of this disease. In chronic gastritis water is best taken between meals; alcoholic stimulants, strong coffee and tea as well as condiments should be forbidden.

In atrophic gastritis, as well as in achylia gastrica, the intestine vicariously digests the food for the stomach. It is therefore well to allow those foods which are easily digested by the intestinal secretion. The food must be thoroughly comminuted and must largely be given in liquid and semisolid form. Of the liquid broths, rice, barley or chicken broth are especially useful; cereals should be well cooked and peas and beans strained and eaten in the purée form. Potatoes and rice should be cooked with milk or broth and eggs should be eaten soft boiled. Meats must be taken only in the most digestible forms, as brains and scraped beef; boiled fish and raw oysters are allowable. When milk is not well digested in this affection, buttermilk, cream and kumiss may be substituted for it. Butter may be eaten on toast or crackers. Small meals should be prescribed and given at frequent intervals. The following diet list may be found serviceable in the treatment of patients affected with achylia gastrica:

Time	Grams	Food	Calories
8 A. M.	200	Milk flavored with cocoa or tea	135
	60	Toast (154) with 20 grams of butter (163)	317
	350	Farina, hominy or rice boiled in milk	440
10 A. M.	200	Milk	135
12 Noon	200	Milk soup	227
	100	Boiled chicken	106
	100	Spinach	166
	100	Mashed potatoes	127
	60	Toast	154
4 P. M.	200	Milk flavored with cocoa	135
7 P. M.	200	Milk	135
	60	Toast	154
	20	Butter	163
	350	Farina in milk	440
			2834

DIET IN ATONY OF THE STOMACH

In patients affected with atony of the stomach great care must be exercised to allow only small quantities of food, which are to be taken at frequent intervals. The quantities of fluids should not be more than one and a half quarts a day, and must be given in small amounts. Large quantities of milk, as are often prescribed, are not advised by the author, especially when the patient is up and about, for the weight of the excessive quantities of fluids overdistends the stomach; however, when at rest in bed, as during a rest cure, milk is better borne in large amounts. In those instances of atony accompanied by hyperacidity a liberal meat diet is to be recommended, and fish and eggs are also allowable, the vegetables, however, should be restricted, and are best given in purée form. Butter in the form of fat is best suited in this condition. In those cases of atony of the stomach associated with hypochlorhydria, the light meats such as chicken and sweetbreads are to be recommended, while vegetables may on the other hand be allowed in larger quantities. For the chronic constipation usually associated with this affection, diet plays an important rôle, and those foods are especially to be advised which increase the intestinal movements. Among these foods are carrots, tomatoes, beans, peas, turnips, as well as raw and stewed fruits, cider, honey, and Graham and rye bread. The following diet list has been utilized by the author in the treatment of atony of the stomach:

Half-past seven o'clock, A. M.:	
60 grams orange juice	132 calories
150 grams of milk with tea	100 calories
100 grams of rice (or other cereal with milk)	127 calories
50 grams of toast	130 calories
20 grams of butter	160 calories
Half-past ten o'clock, A. M.:	
200 grams of milk	135 calories
Half-past twelve o'clock, P. M.:	
100 grams of stewed chicken, chops or steak	106 calories
100 grams of peas (mashed or strained)	318 calories
100 grams of baked potatoes	127 calories
100 grams of stewed apples	53 calories
40 grams of toast or stale bread	102 calories
Half-past three o'clock, P. M.:	
200 grams of cocoa cooked in milk	180 calories
Seven o'clock, P. M.:	
200 grams of milk	135 calories
50 grams of toast	130 calories
30 grams of butter	245 calories
2 soft-boiled eggs	160 calories
Total	2340 calories

DIET IN DILATATION OF THE STOMACH

In dilatation of the stomach fluids are badly borne, and therefore should be given only in small amounts, not over one or two quarts a day. Of the fluids which are to be recommended, milk, cream, buttermilk, tea and coffee and broths may be allowed in small quantities. In order to overcome the intense thirst, the patient may be allowed to suck bits of ice or be given rectal enemas of water or normal salt solution. On account of the general weakened nutrition, nutrient enemata must be prescribed at times. In order to increase the nutritive value of the milk, rice, tapioca or eggs may be added; concentrated beef juice or meat extracts may also be used, as well as broths thickened with rice, barley, tapioca, or eggs. Meat should be allowed only in the most digestible forms such as sweetbreads, chicken, calves' brains and scraped beef. Vegetables must be permitted only in the purée form, and of these carrots, potatoes, peas, and spinach are best. Bread can be taken only stale or toasted or in the form of zwieback. Stewed fruits are of considerable value as they tend to overcome constipation. Butter may be taken in small amounts. Spices and condiments should be avoided. It is important to prescribe small concentrated meals and large quantities of fluids should always be avoided. The following diet list has been found useful by the author in the treatment of dilatation of the stomach:

Half-past seven o'clock, A. M.:	
40 grams of orange juice	88 calories
100 grams of cocoa (cooked in milk)	180 calories
50 grams of toast	130 calories
20 grams of butter	160 calories
Half-past ten o'clock, A. M.:	
100 grams of scraped beef	118 calories
50 grams of toast	130 calories
10 grams of butter	80 calories
Half-past twelve o'clock, P. M.:	
100 grams of lamb chops or chicken or steak	230 calories
100 grams of peas (purée), spinach, asparagus, or carrots	310 calories
100 grams of baked potatoes	127 calories
50 grams of toast	130 calories
10 grams of butter	80 calories
Half-past three o'clock, P. M.:	
100 grams of cocoa in milk	180 calories
Seven o'clock, P. M.:	
2 soft boiled eggs	160 calories
50 grams of toast	130 calories
10 grams of butter	80 calories
Total	2313 calories

DIET IN ULCER OF THE STOMACH

Since Cruveilhier first advised an exclusive milk diet in the treatment of ulcer of the stomach and Brinton first pointed out the advantage of rest in the treatment of this affection, much interest has been manifested in the dietetic treatment of this disease. Many variations in its treatment have been instituted depending largely upon the individual views regarding its etiology. Rosenow's work has in many respects materially altered our views regarding the etiology of ulcer. According to this investigator the cause of this disease is in all probability a hematogenous infection with special strains of streptococci absorbed from some focus of infection. On this account the view is now generally maintained that previous to instituting treatment all sources of focal infection should as far as possible be removed. While, however, infection is a probable etiological factor, it has as yet by no means been proven that it is the sole factor in the causation of ulcer.

Aside from removing all focal infections prophylactically much can be accomplished by means of a carefully selected diet in preventing the onset of ulcer of the stomach. As soon as the first symptoms appear, the patient should be placed upon an exclusive milk diet. The temperature of the food should be regulated, so that it be given not too hot or too cold. Anemia which so commonly accompanies this affection must be overcome and hyperchlorhydria, which apparently bears some etiologic relation to this condition, must as far as possible be reduced; there can be no question but that healing is apparently prevented in some instances by the action of the acid gastric juice. This fact has been demonstrated by Sippy in his method of treatment in ulcer, in that by instituting neutralization of the gastric secretion by the hourly administration of milk and cream as well as by alkalies, the healing of ulcers in many instances is accomplished.

The treatment of ulcer of the stomach must vary according to whether hemorrhage is or is not at hand. When hemorrhage is present absolute rest in bed is indicated; the patient should not be allowed to arise even for purposes of defecation or urination. No nourishment whatever should be allowed by mouth. In robust individuals even nutriment enemata may be omitted. If the patient is in a much enfeebled state, nutrient enemata must be employed and should be continued for from three to five days. Following this, one of three plans of treatment may be followed, *i.e.*, the Leube cure, the Lenhartz cure, or the Sippy cure.

Whenever hemorrhage has not been noted, any of the above cures may be undertaken at once. According to the older plan, the **Leube treatment** was almost constantly followed. This consists of placing the patient at complete rest in bed for fourteen days upon a liquid diet, mainly on milk. Beginning with the third week and continuing during the fourth week, the patient is allowed to recline on a couch. The diet must still consist mainly of milk with the addition of soft food, and after the fourth week, the easily digestible vegetables as potatoes, spinach, peas, carrots, all in the purée form, may be added to the dietary and finally the white meats when well cooked may be allowed.

The Leube diet may easily be carried out by following the dietary tables already noted (page 287). The first dietary should be followed for ten days; the second for the succeeding ten days; the third for eight days. Hot poultices are applied to the abdomen for several weeks during the early part of the treatment and from 200 to 300 c.c. of hot Carlsbad salts are taken morning and evening.

Although milk is the most useful form of food during the first weeks of the rest treatment of ulcer, it occasionally does not agree with the patient, and substitutes must be given in its place. Of these, buttermilk, kefir, kumiss and malted milk may be recommended.

In order to increase the food value of milk, cream may be added, and the following calculation of Strauss may be utilized in order to estimate the increased value:

(*A*)	100 grams of full milk	70 calories
(*B*)	75 grams of full milk with 25 grams of cream	115 calories
(*C*)	50 grams of full milk with 50 grams of cream	185 calories
(*D*)	25 grams of full milk with 75 grams of cream	205 calories
(*E*)	180 grams of cream	250 calories

There are therefore of each—milk, milk and cream, and cream—in the half liter: (*A*) 350 calories; (*B*) 575 calories; (*C*) 925 calories; (*D*) 1025 calories; (*E*) 1250 calories.

Lenhartz has cautioned against the strict abstinence diet in the treatment of ulcer of the stomach even in those instances in which there is hemorrhage. He bases his conclusions on the fact that since ulcer of the stomach is most frequently accompanied by hyperacidity and also by an enfeebled state, it is best to prescribe protein food to easily overcome both conditions.

In the **Lenhartz cure** absolute rest in bed for at least four weeks is maintained. An ice-bag is placed on the abdomen and left on more or less continually for two weeks. On the first day, even though there be hematemesis, 200 c.c. of iced milk are given in teaspoonful doses together with two raw ice-cold beaten eggs. The eggs are beaten up with sugar, and they are kept cold by placing the cup containing them in a dish filled with ice. The milk is increased every day 100 grams and one additional egg is added; on the ninth day the patient is given 1 liter (2 pints) of milk, and the quantity is not increased; on the sixth day raw scraped beef is added, and the quantity is doubled on the following day; on the seventh and eighth days, the patient is given some well-cooked rice and zwieback (softened); and on the tenth day raw ham and butter. (See Lenhartz Diet Scale, page 305.)

According to the author's experience the Lenhartz treatment is to be preferred to the Leube cure in many instances, for a long-continued restriction to a liquid diet is often unnecessary and distressing to the patient and may lead to great loss of flesh, weakness and anemia. This treatment is especially satisfactory in cases accompanied by hemorrhage. Quite satisfactory results are, however, often obtained by the Leube cure, and at times on account of the severe nausea, vomiting and pain, one is forced to follow this form of treatment.

In recent years **Sippy** has evolved a **method** of treating ulcer of the stomach and duodenum which has in a great measure replaced other methods of treatment, and which according to the author's observations in a large number of cases has yielded the more gratifying results. The treatment consists in protecting the ulcer from the acid corrosion until it has healed by shielding it from the corrosive effect of the gastric secretion.

He accomplishes this by maintaining a neutralization of all free hydrochloric acid from early in the morning until late at night. This is affected by frequent feedings and the use of alkalies given frequently.

The patient remains in bed for from three to four weeks. Three ounces of a mixture of equal parts of milk and cream are given every hour from 7 A. M. to 7 P. M. After two or three days, 3 soft boiled eggs, and 9 ounces of well-cooked cereal, with milk and cream mixture every hour, are given each day. Cream soups of various kinds, vegetables, purées and other soft foods may be substituted now and then as desired. Powders of magnesia and soda and calcium carbonate and soda are given between the feedings to neutralize the acid.

DIET AS GIVEN BY LENHARTZ IN HIS ULCER CURE

Day after Hematemesis	Eggs (Number)	Sugar (Grams)	Milk (c.c.)	Beef, Raw Scraped (Grams)	Milk, Cooked with Rice (c.c.)	Zwieback (Grams)	Ham, Raw (Grams)	Butter (Grams)	Calories
1	2	...	200	...	...	...	...	...	280
2	3	...	300	...	...	...	...	...	420
3	4	20	400	...	...	...	...	...	637
4	5	20	500	...	...	...	...	...	779
5	6	30	600	...	...	...	...	...	955
6	7	30	700	35 *b. d.*	...	...	...	...	1135
7	8	40	800	35 *b. d.*	100	...	...	...	1588
8	8	40	900	35 *b. d.*	100	20	...	...	1721
9	8	50	1000	35 *b. d.*	200	40	...	...	2138
10	8	50	1000	35 *b. d.*	300	40	50	20	2478
11	8	50	1000	35 *b. d.*	300	60	50	40	2941
12	8	50	1000	35 *b. d.*	300	60	50	40	2941
13	8	50	1000	35 *b. d.*	300	80	50	40	3007
14	8	50	1000	35 *b. d.*	300	100	50	40	3073

MODIFIED LENHARTZ ULCER DIET OF SEALE HARRIS

First Day.—1 egg, 1½ ounces of cream, 4 ounces of milk. Mix and give one-half ounce every hour from 7 A. M. to 7 P. M. Total calories approximate 300.

Second Day.—2 eggs, 3 ounces of cream, 8 ounces of milk. Mix and give 1 ounce every hour from 7 A. M. to 7 P. M. Total calories approximate 600.

Third Day.—3 eggs, 4½ ounces of cream, 12 ounces of milk. Mix and give 1½ ounces every hour from 7 A. M. to 7 P. M. Total calories approximate 1,200.

Fourth Day.—4 eggs, 6 ounces of cream, 16 ounces of milk. Mix and give 2 ounces every hour from 7 A. M. to 7 P. M. Total calories approximate 1,200.

Fifth Day.—5 eggs, 7½ ounces of cream, 20 ounces of milk. Mix and give 2½ ounces every hour from 7 A. M. to 7 P. M. Total calories approximate 1,500.

Sixth Day.—6 eggs, 9 ounces of cream, 24 ounces of milk. Mix and give 3 ounces every hour from 7 A. M. to 7 P. M. Total calories approximate 1,800.

.. *to Tenth Day.*—6 eggs, 6 ounces of cream, 20 ounces of milk. Mix and give 3 ounces at 8, 9, 10, 11, 12 A. M. and 2, 3, 4, 5, 6 P. M.; and at 7 A. M. and 7 P. M. give two tablespoonfuls of strained oatmeal, 2 ounces of cream, level teaspoonful of sugar, and 1 soft boiled egg; at 1 P. M. give 1 rounded tablespoonful of scraped beef, lightly broiled, 1 heaping tablespoonful of rice, and 3 ounces of milk. Total calories approximate 2,100.

Eleventh to Fourteenth Day.—Breakfast at 7 A. M., and supper, 7 P. M., 2 soft boiled eggs, 1 slice of toast, 1 pat of butter, 3 tablespoonfuls of strained oatmeal or cream of wheat, 3 ounces of cream, and a teaspoonful of sugar. Dinner, at 1 P. M., 2 tablespoonfuls of scraped beef or minced breast of chicken, 2 slices of dry toast, 2 heaping tablespoonfuls of rice, butter and 2 tablespoonfuls of ice cream. 1 egg, 1 ounce of cream and 3 ounces of milk at 9 and 11 A. M. and 3 and 5 P. M. Total calories 2,800.

Fifteenth to Twenty-first Day.—Same as from eleventh to fourteenth days except that the amount of cereal, cream and chicken or beef may be increased, baked Irish potato may be substituted for rice, and gelatin or boiled custard for ice cream. One egg, 1 ounce of cream and 4 ounces of milk should be given at 10 A. M. and 4 P. M. Total calories approximate 3,000.

From the Third to Sixth Week.—The diet should be the same as in the third week except that strained orange juice may be given for breakfast and purées of peas, beans and potatoes may be given for dinner. Soft green vegetables mashed through cloth or sieve may be added. Total calories approximate 3,500.

Note.—The egg, milk and cream mixture should be kept in a covered dish with ice packed around it. The amount for each feeding should be given slowly with a spoon and with the same exactness and regularity as if it were medicine. The scraped beef should be made into a patty and lightly broiled with a little butter and salt. The rice and oatmeal should be cooked for several hours. The dry toast should be in slices about 4 x 4½ inches in size. The crust should be removed and the patient instructed to chew it until it becomes liquid with the saliva.

The after-management of these patients must be carefully carried out. The hourly feeding and alkaline powders must be continued for some weeks after the patient is following his regular occupation. If for any special reason the hourly feeding cannot be maintained the three usual meals must be substituted, and the alkaline powders administered at frequent intervals.

There are a certain proportion of cases of ulcer of the stomach that do not yield to the ordinary treatments. Donkin was the first to direct attention to the fact that excellent results can be obtained in this class of cases by exclusive rectal alimentation. This plan should be carried out for about a period of ten days. The author has found that in administering nutrient enemata far better results are obtained when the enemata are given by the continuous drop method at the rate of 60 drops per hour or less. The enemata may contain milk and grape sugar in salt solution.

Of far greater value in the treatment of these cases is the method of duodenal alimentation as has been advised by Einhorn, attention to which has already been called. (See page 295.)

Einhorn uses as a nutritive medium of from 200 to 240 c.c. of milk and from 15 to 30 grams of lactose every two hours. At times he adds cream, eggs, broth, and oatmeal gruel to increase the caloric value. There can be no question but that relapses are frequently due to indiscretions in diet following the ulcer cure when the patient is no longer under the control of his physician. The patient should be placed upon a carefully regulated diet free from acids and indigestible foods; intermediate feedings should be prescribed and alkalies given for some months following the cure. When possible it is the author's habit to have his patients report for reëxaminations at periods of from three to four months for a year or more so as to determine the ultimate result of the treatment. On page 310 is presented a diet list which the author's patients follow as an after-treatment.

Sippy Diet Cure Modified by Friedenwald and Ruhräh

(*From "Diet in Health and Disease"*)

Hour	1st to 5th Days	6th Day	7th to 8th Days
A. M.			
7.00	Milk and Cream *	Soft Egg, Milk and Cream	Milk and Cream
8.00	Milk and Cream	Milk and Cream	Milk and Cream, Soft Egg
9.00	Milk and Cream	Milk and Cream	Milk and Cream
10.00	Milk and Cream	Milk and Cream	Milk and Cream
11.00	Milk and Cream	Milk and Cream	Milk and Cream
12.00	Milk and Cream	Milk and Cream	Milk and Cream
P. M.			
1.00	Milk and Cream	Milk and Cream	Cereal, Egg, Milk and Cream
2.00	Milk and Cream	Milk and Cream	Soft Egg, Milk and Cream
3.00	Milk and Cream	Milk and Cream	Milk and Cream
4.00	Milk and Cream	Milk and Cream	Milk and Cream, Cereal
5.00	Milk and Cream	Milk or Cocoa, Soft Egg	Milk and Cream
6.00	Milk and Cream	Milk and Cream	Milk and Cream
7.00	Milk and Cream	Milk and Cream	Milk and Cream

Hour	9th to 10th Days	11th to 14th Days	15th Day
A. M.			
7.00	Cereal, Milk and Cream	Soft Egg, Cereal, Milk and Cream	Egg, Cereal, Milk and Cream
8.00	Milk and Cream	Milk and Cream	Milk and Cream
9.00	Milk and Cream	Milk and Cream	Milk and Cream
10.00	Milk and Cream	Milk and Cream	Milk and Cream
11.00	Milk and Cream	Milk and Cream	Milk and Cream
12.00	Milk and Cream	Milk and Cream	Milk and Cream
P. M.			
1.00	Cereal, Egg, Cocoa	Egg, Cocoa, Custard	2 Eggs, Cocoa, Milk Toast
2.00	Milk and Cream	Milk and Cream	Milk and Cream
3.00	Milk and Cream	Milk and Cream	Milk and Cream
4.00	Milk and Cream	Milk and Cream	Milk and Cream
5.00	Milk Toast, Egg and Cocoa	Milk Toast, Egg and Cocoa	Milk Toast, Egg and Cocoa
6.00	Milk and Cream	Milk and Cream	Milk and Cream
7.00	Milk and Cream	Milk and Cream	Milk and Cream

Sippy Diet Cure Modified by Friedenwald and Ruhräh (Cont'd)

(*From "Diet in Health and Disease"*)

Hour	16th Day	17th to 18th Days	19th Day
A. M.			
7.00	Egg and Cereal, Milk and Cream	Soft Egg, Cereal, Cocoa	Milk and Cream
8.00	Milk and Cream	Milk and Cream	Milk and Cream
9.00	Milk and Cream	Milk and Cream	Chicken Broth
10.00	Milk and Cream	Milk and Cream	Milk and Cream
11.00	Milk and Cream	Milk and Cream	Milk and Cream
12.00	Milk and Cream	Milk and Cream	Milk and Cream
P. M.			
1.00	Cream, Milk Toast, Egg, Vanilla Ice Cream	Minced Chicken, Milk, Milk Toast, Vanilla Ice Cream	Minced Chicken, Cocoa, Dry Toast, Cocoa, Dry Toast, Vanilla Ice Cream
2.00	Milk and Cream	Milk and Cream	Milk and Cream
3.00	Milk and Cream	Milk and Cream	Milk and Cream
4.00	Milk and Cream	Milk and Cream	Milk and Cream
5.00	Milk Toast, Egg, Cocoa	Milk Toast, Egg, Cocoa	Milk Toast, Egg, Cocoa
6.00	Milk and Cream	Milk and Cream	Milk and Cream
7.00	Milk and Cream	Milk and Cream	Milk and Cream

Hour	20th Day	21st Day	22nd Day
A. M.			
7.00	2 Eggs, Cocoa, Slice Dry Toast, Butter	2 Eggs, 1 Slice Toast, Butter	Milk and Cream
8.00	Milk and Cream	Milk and Cream	Milk and Cream
9.00	Milk and Cream, Egg	Milk and Cream, Egg	Cereal, Milk and Cream, Egg
10.00	Milk and Cream	Milk and Cream	Milk and Cream
11.00	Broth	Milk and Cream	Milk and Cream
12.00	Milk and Cream	Milk and Cream	Milk and Cream
P. M.			
1.00	Minced Chicken, 1 Slice Dry Toast, Butter, Cocoa, Spinach	1 Broiled Lamb or Mutton Chop, Dry Toast, Cocoa, Butter Asparagus or Baked Potato	Chop or Minced Chicken, Dry Toast, Strained Vegetable, Baked Potato, Cocoa, Butter
2.00	Milk and Cream	Milk and Cream	Milk and Cream
3.00	Broth	Milk and Cream or Milk and Egg	Milk or Cream
4.00	Milk and Cream	Milk and Cream	Milk and Cream
5.00	Toast, Cocoa, 2 eggs, Cereal and Milk	Toast, Cocoa, 2 eggs, Cereal and Milk	Stewed Fruit or Baked Apples, 2 Eggs, Cereal, Milk Toast, Cocoa
6.00	Milk and Cream	Milk and Cream	Milk and Cream
7.00	Milk and Cream	Milk and Cream	Milk and Cream

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* Milk and Cream, each 1½ ounces, throughout the table.

Modified Sippy Diet Followed in Some Cases of Peptic Ulcer

(*Milk and Cream, Equal Parts,* 1½ *Ounces Every Hour from* 7.00 A. M. *to* 7.00 P. M.)

Nine to Twelve Days

Hours A. M.	Hours P. M.	Medicines and Food	Quantity
7.30	1.30	Heavy calcined magnesia	Grains x
9.00	3.30-5.30	Sodium bicarbonate	Grains x
11.30	7.30-9.30		
	12.30-2.30	Calcium carbonate	Grains x
8.30	4.30-6.30	Sodium bicarbonate	Grains xx

Three to Four Days Following

7.00	3.00	Milk and cream	āā ounce
11.00	7.00		
9.00	1.00-5.00	Milk and egg or cocoa	
7.30	3.30	Heavy calcined magnesia	Grains x
11.30	7.30	Sodium bicarbonate	Grains x
9.30	1.30	Calcium carbonate	Grains x
	5.30-9.30	Soda bicarbonate	Grains x

Four to Five Days Following

7.00-11.30	3.00-7.00	Milk and cream	āā ounces iss
9.00	....	Oatmeal, egg and cocoa	
	1.00-5.00	Milk toast, egg and cocoa	
7.30	3.00	Heavy calcined magnesia	Grains x
11.30	7.30	Sodium bicarbonate	Grains x
9.30	1.30	Calcium carbonate	Grains x
	5.30	Sodium bicarbonate	Grains x

To be Followed for Some Weeks

7.00	3.00-7.00	Milk and cream	āā ounces iss
11.00	....	Milk and egg or cocoa	
9.00	1.00-5.00	Soft diet without acids	
7.30	3.30	Heavy calcined magnesia	Grains x
11.30	7.30	Sodium bicarbonate	Grains x
9.30	1.30	Calcium carbonate	Grains x
	5.30-9.30	Sodium bicarbonate	Grains x
		As required, additional sodium bicarbonate	

Diet List Following an Ulcer Treatment

(*In Addition to the Usual Meals Intermediate Feedings Should Be Taken*)

MAY TAKE

Soups:

- Clam
- Mutton
- Barley
- Rice
- Vermicelli
- Cream
- Potato
- Pea
- Celery
- Asparagus

Meats (minced):

- Boiled
- Broiled
- Lamb
- Lamb Chops
- Mutton
- Mutton Chops
- Brains
- Sweetbreads

Poultry:

- Boiled
- Broiled
- Roasted
- Chicken
- Turkey
- Squab

Bread:

- Stale
- Toasted
- Pulled
- Wheat
- Zwieback
- Crackers

Fish (minced):

- Boiled
- Baked
- Broiled
- Bluefish
- Bass
- Haddock
- Halibut
- Trout
- Mackerel
- Oysters
  - Raw
  - Steamed
  - Stewed
  - Broiled
- Clams
  - Broth
  - Raw
  - Steamed

Eggs:

- Raw
- Soft Boiled
- Poached
- Omelet
- Scrambled
- Shirred

Cereals:

- Rice
- Cracked Wheat
- Cornmeal
- Barley
- Oatmeal
- Cream of Wheat
- Farina
- Hominy
- Grits
- Shredded Wheat

Milk:

- Whole
- Skimmed
- Whey
- Curd
- Junket
- Matzoon
- Kumiss
- Kefir
- Cream
- Pasteurized
- Buttermilk

Ice Cream:

- Vanilla

Farinaceous Food

- Cornstarch
- Tapioca
- Sago
- Vermicelli

Butter:

Vegetables:

- Asparagus
- Spinach
- Cauliflower
- Squash
- Watercress
- Potatoes
  - Mashed
  - Baked
- Carrots
- Artichoke
- Lima Beans
- Lentils
- Peas
- String Beans

- Pears
  - Stewed
- Peaches
  - Stewed
- Prunes
- Apples
  - Stewed
  - Baked

Beverages:

- Barley Water
- Oatmeal Water
- Rice Water
- Albumin Water
- Tea (weak)
- Coffee
- Cocoa

Desserts:

- Puddings
- Rice
- Blanc Mange
- Cornstarch
- Bread
- Tapioca
- Cup Custard

Mineral Waters:

- Vichy
- Hawthorne
- Lithia
- Apollinaris
- White Rock
- Poland

ARTICLES OF DIET TO BE FORBIDDEN

- Candies
- Salads
- Fried Foods
- Alcoholic Stimulants
- Sweet Potatoes
- Strong Tea
- Strong Coffee
- Cabbage
- Salted Fish
- Smoked Fish
- Sardines

- Salmon
- Preserved Fish
- Beef
- Pork
- Veal
- Crabs
- Rice Soups
- Liver
- Kidneys
- Duck
- Corn

- Goose
- Potted Meats
- Corned Meats
- Stews
- Hashes
- Sausage
- Twice Cooked Meats
- Celery
- Beets
- Radishes
- Hot Cakes

- Hot Bread
- Berries
- Bananas
- Melons
- Oranges
- Grapefruit
- Lemons
- Preserves
- Pastry
- Pies
- Nuts
- Tomatoes

Smithies' Ulcer Diet

(Based upon established clinical, histopathological and physiological facts)

Days 1 *to* 7. *(Time Varies According to Patient's Condition.)*

By Mouth: One-half ounce warm water hourly when awake. Patient chews paraffin wax for fifteen minutes at least once in two hours. Juice of sweet orange or grapefruit occasionally.

By Rectum: Nutrient enema consisting of 50 per cent. alcohol 1 ounce, glucose sirup 1 ounce, and normal salt solution 6 ounces every four hours. The enemata are preceded by a cleansing irrigation of the colon with normal salt solution. They are given at body temperature by the drop method at the rate of from 30 to 60 drops per minute. Calories daily approximately 1000. During the first two days, tincture of opium, minims 10, is added to each second enema.

Days 3 *to* 14. *(Case of Average Severity.)*

By Mouth: From 4 to 6 ounces of water gruel at temperature of 100° F. (37.8° C.). The gruel is taken slowly through a glass tube. Gruels are made from rice, cream of wheat, oatmeal, sago, cornmeal, milk, macaroni, and vermicelli, rusks, potato, asparagus, cauliflower, beans, peas, and boiled onion. They are strained before feeding. Flavoring with coffee, chocolate, vanilla, caramel, etc., renders the cereal gruels palatable and their administration easier. To the vegetable gruels small quantities of arrowroot or cornstarch are added to secure a thin emulsion. Before each feeding, paraffin wax is chewed for five minutes. Warm water or sweet orange or grapefruit juice are allowed as desired, but never in greater quantity than 1 ounce at a time.

By Rectum: During the first two days of mouth feeding, two alcohol glucose-saline nutrient enemata are given. During the second two days, one such nutrient enema is administered. After the fourth day of mouth feeding no rectal feedings are given in the average case. Calories approximately 800.

Days 14 *to* 21

Half-past six o'clock, A. M.: A glass of hot water and 1 teaspoonful of non-effervescent sodium phosphate.

Half-past seven o'clock, A. M.: One ounce of sweet orange or grapefruit juice, two ounces of thin cream of wheat, or farina, or well-cooked rice, or cornmeal, 2 ounces of skimmed, parboiled milk, may be taken with cereal, and if desired a small quantity of powdered sugar used. One zwieback with a thin layer of fresh butter, 4 ounces of parboiled skimmed milk, containing half volume of lime-water, served warm, and flavored with coffee, cocoa, caramel, or vanilla.

Half-past nine o'clock, A. M.: Six ounces of thin water gruel from cereals or fresh vegetables, strained and served hot, 1 rusk or zwieback, or dry toast.

Half-past eleven o'clock, A. M.: Four ounces of malted milk, whipped egg with parboiled milk, cornstarch pudding, simple custard lightly cooked.

Half-past twelve o'clock, P. M.: Six ounces potato, pea, bean, or asparagus purée (strained), or vegetable broth; 4 ounces of salisbury steak (moderately well cooked) to chew; 2 ounces (cooked weight) of thin rice, sago, tapioca, or cornstarch pudding made with parboiled milk and egg yolk; 2 ounces of parboiled milk and small quantity of pulverized sugar may be eaten with the pudding; 1 rusk or zwieback, 6 ounces of parboiled milk and quarter volume of lime-water flavored to taste.

Four o'clock, P. M.: Four ounces of water gruel from cereals, 1 very soft poached egg, 1 rusk or zwieback, 4 ounces of hot Vichy water.

Six o'clock, P. M.: Four ounces of whipped egg, 2 rusks or zwieback, 6 ounces of malted milk (thin), flavored to taste, or cereal water gruel or parboiled milk and quarter volume of lime-water gruel.

Nine o'clock, P. M.: Six ounces of water cereal gruel or 4 ounces of malt marrow, 2 Graham crackers. Calories approximately 1500.

Days 21 *to* 42

Half-past six o'clock A. M.: Two teaspoonfuls of phosphate of soda in a glass of hot water.

Eight o'clock, A. M.: Juice of 1 sweet orange or half sweet grapefruit, or boiled prunes passed through fine colander; 2 ounces (cooked weight) of thin cereals (cream of wheat, farina, oatmeal, cornmeal), 2 ounces of skimmed milk and small amount powdered sugar, 1 soft poached egg, 2 zwieback, 2 rusks or 2 thin slices of well-toasted Graham bread, 1 pint of hot skimmed milk, a quarter volume of lime water flavored to taste (cocoa, vanilla, etc.).

Ten o'clock, A. M.: One pint of hot parboiled whole milk and fifth volume of lime-water, 2 rusks or Graham crackers.

Half-past twelve o'clock, noon: Four ounces of creamed soup from vegetables, strained, 6 ounces rare meat to chew, 4 ounces well-mashed potato (mealy inside), or carrot, peas, beans, cauliflower, Brussels sprouts, or asparagus (all vegetables passed through a strainer and served with 15 grams of butter; 4 ounces (cooked weight) of pudding from rice, cornstarch, sago, tapioca, cream of wheat, or farina, or 4 ounces of custard, pulp of sweet orange, grapefruit, or prune whip, or chew 6 ounces of watermelon or cantaloupe, half pint of hot skimmed milk.

Half-past three o'clock, P. M.: One hundred fifty cubic centimeters of hot whole milk and quarter volume of lime-water or 150 c.c. of malted milk or weak cocoa.

Half-past six o'clock, P. M.: Two rusks of zwieback or 2 slices of well-toasted Graham bread, 2 very soft poached eggs, 100 grams of sweet-apple sauce or 1 baked apple (omit skins), or juice of sweet orange or half of grapefruit or chew 6 ounces of melon, 1 pint of skimmed milk hot.

Nine o'clock, P. M.: Two hundred fifty cubic centimeters of whole parboiled milk and quarter volume of lime water or 250 c.c. of malted milk, hot. Calories for twenty-four hours approximately 2000.

General Diet after Three Months

(If distress, patient should go back to 7 to 21 day diet)

Seven o'clock, A. M.: One pint of skimmed milk and half gill of cream.

Nine o'clock, A. M.: Two pieces of toast without butter, juice of one sweet orange or grapefruit or ripe melon or apple sauce or baked apple (do not eat skin) or marmalade, 1 dish of well-cooked cereal (oatmeal, farina or cream of wheat), 2 very soft poached eggs, 2 cups of hot sweetened water. The water may be made more palatable by flavoring with cocoa, tea, coffee or cream.

Eleven o'clock, A. M.: One cup of bouillon (two cubes), two Graham crackers.

One o'clock, P. M.: This should be the heavy meal of the day. It may consist of meat (rare beef or rare hamburger steak, lamb or white meat of fowl), fish (never fried), oysters, well-cooked spinach, cauliflower, carrots, squash, peas (hulled), string beans, Brussels sprouts, baked or mashed potatoes (in moderation), rice with gravy, simple puddings made from cereals, cornstarch, gelatin, well-cooked fruit sauces, simple cakes, no white bread (all bread should be made from dark flour and should be at least one day old), 1 pint of skimmed milk taken hot.

Four o'clock, P. M.: One glass of hot peppermint water (20 drops of "essence" of peppermint to the glass, sweeten to taste and drink slowly), two Graham crackers.

Seven o'clock, P. M.: A light lunch consisting of vegetable soup, simple salad, toast, soft eggs, and plain puddings or cake, with or without ripe cooked fruit sauces, 1 pint of hot skimmed milk.

Bedtime: One glass of malt marrow, malted milk or hot skimmed milk. Calories approximately 3500 for twenty-four hours.

DIET IN CANCER OF THE STOMACH

In those cases in which the cardia of the stomach is involved the diet should be of such a nature as not to cause any further irritation of the strictured area. In consequence food should be largely of the liquid variety, and should be best given in the form of milk. This form of food may be made more nutritious by the addition of cream, or eggs, cocoa and coffee, and buttermilk may be also taken. When great difficulty in deglutition arises, gastrostomy should be performed, and feeding maintained through the new opening. In addition the use of nutrient enemata is indicated.

When other parts of the stomach are involved, milk still remains a most useful form of food. In addition the more digestible foods such as sweetbreads or boiled chicken are to be recommended, but inasmuch as there is usually a marked distaste for meat in this disturbance, fish and eggs may have to be substituted. The light vegetables as baked or mashed potatoes, beans, peas, and carrots in the purée form as well as asparagus and spinach may also be taken.

On account of the thirst, iced milk, alone or flavored with tea, cocoa or coffee may be allowed. It is sometimes imperative in this affection to assist the general nutrition by means of nutrient enemata. Inasmuch as but little can be expected as the result of food in cancer of the stomach, it is unwise to restrict the diet too greatly in this affection or to urge upon the patient the necessity of taking very large quantities. The author has found the following diet table useful in some cases of cancer of the stomach:

Diet Table for Cancer of the Stomach

Half-past seven o'clock, A. M.:	
20 grams of milk with tea or coffee	135 calories
2 soft boiled eggs	160 calories
50 grams of toast	130 calories
10 grams of butter	80 calories
Half-past ten o'clock, A. M.:	
40 grams of orange juice	88 calories
30 grams of toast	77 calories
10 grams of butter	81 calories
Half-past twelve o'clock, P. M.:	
Bouillon	0 calories
100 grams of squab (or calves' brains, sweetbread or chicken)	100 calories
100 grams of mashed potatoes	127 calories
20 grams of toast	52 calories
100 grams of spinach or asparagus	166 calories
Four o'clock, P. M.:	
200 grams of cocoa with milk	180 calories
Seven o'clock, P. M.:	
200 grams of milk	180 calories
100 grams of boiled rock fish	80 calories
50 grams of toast or cereal	130 calories
10 grams of butter	80 calories
Total	1846 calories

DIET IN HYPERCHLORHYDRIA AND HYPERSECRETION

In the treatment of these affections care must be exercised to regulate the diet so as to prevent an increase in the acid of the gastric secretion. This can be best accomplished at least in a large measure by a regulation of the diet. Inasmuch as it has now been definitely determined that the symptoms ordinarily attributed to the increase in acid are not entirely due to this condition, but to other factors as well, *i.e.,* vagotonia, much can be accomplished by diet alone in overcoming these symptoms even though the acid content may still remain high. It is necessary to prohibit all irritating foods, such as condiments and spices, acids, all alcoholic beverages and hard substances, such as nuts, which are apt to irritate the stomach. Food should not be eaten too hot or too cold and much be thoroughly masticated. The diet should consist mainly of proteins, since this class of foods combine with and tend to neutralize the excess of hydrochloric acid. Among such foods are milk, eggs, meat and fish. On account of the large amount of extractives in meat, which stimulate the flow of the gastric secretion, meat can be allowed only when well cooked so as to remove the extractives. On the other hand, carbohydrates may be given only in the most digestible form and in but small amounts.

Farinaceous foods are not so easily digested in this condition, and must be allowed in the most digestible forms. Of the vegetables to be recommended in this affection only the most digestible can be allowed, as mashed or baked potatoes, spinach, carrots, peas and beans, and then only in the purée form. Inasmuch as fats have a tendency to decrease the acidity of the gastric secretion, the more digestible forms may be allowed as butter, cream and olive oil. The alkaline mineral waters are useful in lessening the gastric acidity, and may be given as Vichy, Apollinaris and Seltzer.

A salt-free diet has been recommended by some writers in the treatment of these affections. There can be no question but that a restricted salt diet is of advantage in the treatment of hyperacidity and hypersecretion. In aggravated cases of nervous hyperchlorhydria a purely vegetable or lacto-vegetable diet has been recommended.

According to the author's experience in dealing with patients taking but little nourishment, it is best to prescribe nourishment at frequent intervals; if, however, large meals are consumed it is advisable to permit but three meals a day. The author has utilized the following diet list to advantage in cases of hyperchlorhydria and hypersecretion:

Half-past seven o'clock, A. M.:	
200 grams of milk flavored with tea	135 calories
2 soft boiled eggs	160 calories
60 grams of toast	154 calories
40 grams of butter	326 calories
Ten o'clock, A. M.:	
50 grams of scraped beef	60 calories
50 grams of toast	130 calories
Half-past twelve o'clock, P. M.:	
100 grams of chicken (or chops or sweetbreads or steak)	106 calories
100 grams of asparagus (or spinach, carrots, peas or beans)	185 calories
100 grams of stale wheat bread	256 calories
100 grams of apple sauce	53 calories
100 grams of milk	100 calories
100 grams of crackers	188 calories
Seven o'clock, P. M.:	
100 grams of baked trout	106 calories
100 grams of milk	67 calories
20 grams of butter	77 calories
60 grams of toast	153 calories

DIET IN NERVOUS GASTRIC AFFECTIONS

Nervous Anorexia and Vomiting.—In nervous anorexia and vomiting it is of prime importance to isolate the patient from his family and in aggravated forms he should be required to remain in bed and given a complete rest cure. Milk in gradually increasing amounts should be given at first, which should soon be followed by other liquids and semisolid foods. Nourishment must be prescribed in small quantities and given at frequent intervals. If insufficient food is taken the patient must be fed by means of nutrient enemata, or, better still, duodenal alimentation should be practiced. The author is convinced that the lives of many affected with these conditions under his care have been saved by means of duodenal feeding. As soon as the patient is able to retain a small amount of nourishment, scraped beef, eggs, rice and toast are especially valuable. At times the most digestible forms of food are vomited in these affections while the most indigestible are retained, so that no attempt should be made to maintain a too vigorous diet.

Diet in Nervous Dyspepsia.—In this condition it is also important not to diet the patient too rigidly. The patient's strength should be built up and on this account milk should be given in large quantities; if milk is not well borne, buttermilk or kumiss may be substituted for it. As it is important to properly nourish the patient it is advisable to allow any food the patient can digest. Coffee, tea and alcoholic stimulants must be avoided. In aggravated forms of nervous dyspepsia a rest cure is indicated.

DIET IN INTESTINAL DISEASES

The diet is quite as important in the treatment of intestinal diseases as in the treatment of gastric affections. In many instances a cure can be brought about by diet alone, even without the use of drugs. The process of digestion in the intestine is extremely complicated which renders the question of the digestibility of food in the intestine often difficult to determine. The diet should be of such a character as not to produce annoying symptoms and must be easily absorbed. The table given by Rubner (see page 316) presents the degree of absorption of various foods in the intestine:

It is evident that, notwithstanding the large proportion of protein matter in certain foods, its absorbability is so slight that the nutritive value is much less than that of foods containing far less protein. The absorbability depends to a large degree upon the method of preparing the food, as, after mashing and straining, vegetables become much more easily absorbable, inasmuch as some of the cellulose is removed. The digestibility of various foods in the intestine differs in different individuals, which makes it impossible to formulate exact rules for all cases. The diet must be regulated personally for each individual. Foods may be divided into three classes, according to their effect on the peristaltic movements of the intestine: Foods which produce constipation; those which produce a laxative effect; and those which have no effect either way. Among the constipating foods are red wines, cocoa, tea, rice, tapioca, barley, macaroni. In the class of laxative foods are fruits, tomatoes, cabbage, cider, beer, buttermilk, and alkaline carbonated waters, while those foods having no especial effect on the movements of the bowels comprise meat, fish, eggs and wheat breads. However, while in some patients certain foods may have a laxative effect they may have a constipating effect in others, so that no exact general rules can be formulated, but the tendencies of each person must be especially taken into account, a point that cannot be too strongly insisted upon. In aggravated forms of intestinal affections rectal alimentation may have to be prescribed or duodenal feeding be resorted to.

RUBNER'S ABSORPTION TABLE

Foodstuffs	Weight of Same in Grams		Relative Percentage Absorbed				
	Fresh	Dried	Dried Substance	Protein	Fat	Carbohydrate	Ash
Meat	984	375	95	97	95	...	82
Eggs	984	247	95	97	95	...	82
Milk	2470	315	92	94-99	95-97	100	54
Milk and Cheese	2490	420	94	96	97	100	74
White bread ..	860	753	95	81	..	99	93
Black bread ..	1360	765	85	68	..	89	64
Macaroni	695	626	96	83	94	99	76
Indian corn ..	750	641	93	85	83	97	70
Corn and Cheese	....	780	96	93	91	96	81
Rice	638	552	96	80	93	99	85
Peas	600	521	91	83	..	96	68
Potatoes	3078	819	91	68	96	92	84
Cabbage	3830	406	85	82	94	85	81
Carrots	2506	352	79	61	94	82	76

The Schmidt diet test will frequently determine the digestibility of food in the intestine; it will also determine whether carbohydrate fermentation or protein putrefaction is at hand.

The diet may be so varied as to prevent certain undesirable bacterial flora from multiplying to a great extent, and frequent changes in diet are beneficial in combating the rapid growth of any one type of bacteria. According to Hull and Rettger milk and lactose form the most effective means of overcoming the putrefying types of microörganisms.

Antiputrefaction Diet.—Milk forms the most valuable food containing protein and yet it acts in an antiseptic manner. If it be badly borne it may be made more easily digestible by the addition of rice, citrate of soda, lime water or milk of magnesia. It may also be given in the form of milk soups or mushes. Occasionally kefir or kumiss may be substituted for it. Gelatin is also valuable as it tends to inhibit bacterial growth in the intestine and is therefore antiputrefactive; when the patient has improved, other forms of food may be cautiously prescribed. Of these zwieback, crackers, butter and cereals are especially valuable.

Antifermentative Diet.—An antifermentative diet is utilized to overcome as far as possible the fermentation produced by carbohydrates. This condition is ordinarily recognized by the passage of semisolid stools which are spongy and highly acid. In the treatment of these cases the carbohydrate food must be greatly reduced in amount or entirely forbidden until the fermentative changes disappear. At first the diet must consist of clear broth with toast or stale bread and then cereals may be allowed in addition to milk given cautiously; finally solids may be permitted in the form of chicken and broiled meats.

Diet in Intestinal Dyspepsia.—In intestinal dyspepsia the diet should consist of moderate quantities of food, given at frequent intervals. At first only liquids should be taken, such as weak tea, milk, malted milk, and bouillon, and the diet gradually increased, after four or five days, to soft food, and finally solid food in the form of sweetbreads, broiled chops, boiled fish, soft-boiled eggs, spinach, asparagus and stewed fruits may be allowed.

The following diet list taken from Friedenwald and Ruhräh's "Diet in Health and Disease" has been used by the author in the treatment of intestinal dyspepsia:

Eight o'clock, A. M.:	
150 grams of milk with tea	101 calories
1 soft-boiled egg	80 calories
60 grams of toasted wheat bread (155) with 20 grams of butter (163)	218 calories
Ten o'clock, A. M.:	
Scraped beef, sandwich (100 grams of scraped beef [118], 50 grams of wheat bread [178])	296 calories
Twelve o'clock noon:	
Bouillon with 5 grams of Armour's soluble beef	10 calories
100 grams of broiled chicken, or 100 grams of broiled steak (209), or 100 lamb chop (220)	106 calories
50 grams of mashed potatoes, or 100 grams of spinach (166)	64 calories
100 grams of apple sauce	63 calories
Three o'clock, P. M.:	
50 grams of wheat bread, stale or as toast	130 calories
200 grams of milk	135 calories
200 grams of milk with rice	253 calories
1 soft-boiled egg	80 calories
Seven o'clock, P. M.:	
109 grams of wheat bread and 50 grams of butter	666 calories
Total	2222 calories

Diet in Acute Intestinal Catarrh.—The regulation of the diet in acute intestinal catarrh is of the greatest importance. The patient should be kept in bed and fed exclusively on liquid food for a few days, which should consist of clear broth, weak tea and cocoa and egg albumen. Milk should not be allowed in this affection. The thirst may be relieved by allowing small bits of crushed ice to melt in the patient's mouth or carbonated waters may be permitted in small amounts. As soon as the patient is relieved of his pain and discomfort, soft and light solid food consisting of cereals, toast, stewed chicken, soft-boiled eggs and baked potatoes may be allowed. Raw fruit, fatty and acid foods and the heavy vegetables must be avoided for a considerable period of time after the patient is well.

Diet in Chronic Intestinal Catarrh.—The treatment of this condition depends largely as to what portion of the intestine is involved; that is, whether the affection is associated with chronic constipation or chronic diarrhea or whether diarrhea alternates with constipation. In those instances in which there is chronic constipation it is important to prescribe such foods as will stimulate intestinal peristalsis; astringents must be avoided. Those foods which are of most value are rye and Graham bread, buttermilk, cider, honey, fruit (raw and stewed), such vegetables as cabbage, and sauerkraut as well as salads. Fats are especially useful and olive oil is of great value in this affection. Salted foods tend to increase intestinal movements and such foods as caviare and herring are to be recommended. Sugar is of considerable value in this affection, and milk sugar is especially useful. Cold water is also beneficial when taken in the morning on an empty stomach.

When diarrhea exists the patient should be kept quiet in bed and food given in small quantities at frequent intervals. The foods of most value in this condition are broths containing barley and rice, stewed chicken, soft-boiled eggs, boiled fish, toast, baked and mashed potatoes, cocoa, boiled milk and port wine. At times milk disagrees even though it be boiled, and must be prohibited.

When diarrhea alternates with constipation it is important to treat the most pronounced symptom whether this be diarrhea or constipation and to vary the diet frequently according to the condition at hand.

Mineral waters are of considerable value in the treatment of chronic intestinal catarrh. When constipation is present the Marienbad and Saratoga (Hawthorn and Congress) waters are most useful, while when diarrhea exists Carlsbad and Vichy are most beneficial.

Diet in Dysentery.—The diet in acute dysentery is similar to that prescribed in acute intestinal catarrh. The patient is kept quiet in bed on liquid foods consisting mainly of broths, egg albumen and weak tea. As he improves semisolids are allowed, such as milk toast, rice cooked in milk or broth, and solid food should be given only when the disorder has almost entirely disappeared.

In chronic dysentery food should be allowed in small quantities and should be given at frequent intervals. All indigestible food must be avoided. The diet is similar to that prescribed under the head of chronic intestinal catarrh.

Diet in Ulcers of the Intestines.—DUODENAL ULCER.—The same dietetic regulations must be observed in the treatment of duodenal ulcers as has already been described for the treatment of gastric ulcers. When there is hemorrhage the patient must be kept quietly in bed and fed by means of nutrient enemata. After a week or so the usual ulcer cure must be inaugurated.

Other Forms of Intestinal Ulcers.—In other forms of intestinal ulcerations, whether these be dysenteric, tuberculous, or syphilitic, the diet must be very digestible and nonirritative. Such foods as milk, broths, eggs, cereals, toast, sweetbreads and stewed chicken and baked and mashed potatoes may be allowed.

Diet in Acute and Chronic Intestinal Obstruction.—In acute intestinal obstruction operation is usually indicated. Before operation is undertaken the patient must be kept quiet in bed and must abstain from all nourishment; thirst may be quenched by allowing small bits of ice to melt in the mouth. In chronic intestinal obstruction food should be administered only in liquid or semisolid form. The patient must avoid all indigestible food, especially those forms leaving a large residue in the bowels, such as the heavy vegetables and fruits. The foods which are to be recommended in this condition are broths, milk, eggs, broiled meats, chicken, cereals, stale bread and toast.

Diet in Appendicitis.—Inasmuch as surgical intervention is usually indicated in this affection, the dietetic treatment can be carried out only until this procedure is undertaken, or when operation cannot be performed. The patient is placed at rest in bed ordinarily upon liquid food, as albumin water, weak tea, thin broths and moderate quantities of milk diluted with lime water. After the acute symptoms have abated, the diet may be increased, eggs, scraped beef, stewed chicken, squab, and toast may be added to the dietary, and finally light vegetables in the purée form. In cases of appendicitis in which operation must be postponed on account of the condition of the patient, Ochsner advises the following plan of treatment: In all cases of acute appendicitis all food is prohibited by mouth, and all cathartics avoided. Whenever nausea or vomiting exists gastric lavage is undertaken. In mild cases of this affection the patient is allowed to rinse his mouth with cold water, and drink a very small sip of hot water, but in the more aggravated forms, neither hot nor cold water must be taken for several days until the acute attack has subsided. If the nausea persists, lavage of the stomach may be practiced at four-hour intervals in order to remove such substances as have been regurgitated from the bowels into the stomach. The patient is supported by nutrient enemata, and when no water is given by mouth, normal salt saline solution may be administered per rectum. When the pain has subsided for a number of days, one to four ounces of beef tea may be allowed at two-hour intervals. In a few days one of the predigested foods must be substituted, and still later equal parts of milk and lime water, thin liquids in general, then light diet, and finally when the patient has fully recovered full diet may be allowed.

Diet in Mucomembranous Catarrh of the Intestines.—Authors vary as to the character of diet to be recommended in this affection. Fleiner recommended a simple diet; Von Noorden, a very coarse diet; and the author's experience has convinced him that a moderate diet is best, and he therefore prescribes a nutritious mixed diet, which is not too coarse. With such a diet the bowel movements become normal and the amount of mucus secreted is lessened. Von Noorden's diet consists largely of Graham bread, 250 grams a day, in addition to a great variety of leguminous vegetables, including the husks and vegetables containing much cellulose, and fruit with small seeds and thick skinned, like currants, gooseberries and grapes, besides large quantities of fat, particularly butter and bacon. The beneficial effect of this diet is chiefly due to the amount of cellulose it contains. The cellulose undergoes decomposition in the intestines, and thus softens the movements. In connection with the diet, mineral waters are utilized; those containing sodium chlorid are especially useful and particularly the waters of Wiesbaden and Kissingen.

Diet in Enteroptosis.—Much can be accomplished in this condition by means of a carefully regulated diet. Favorable results are frequently obtained by means of rest cures, the patient being required to remain in the recumbent posture with the foot of the bed elevated a long period of time and to consume large quantities of food. Ordinarily patients affected with this disorder should be required to rest after meals, and the diet should be as nourishing as possible, and must contain considerable proportions of fatty foods. Milk is extremely useful in this affection and when cream is added its caloric value is greatly increased. After milk has been consumed for several weeks soft and solid food may be allowed, such as cereals, soft-boiled eggs, meats of various kinds, fish and the more digestible vegetables. When constipation is very marked, foods that increase intestinal peristalsis are especially indicated, among which are buttermilk, cider, grape juice, fruits and honey. When forced feeding is required, as in those instances in which the patient has lost much flesh, cream may be given in from one-half to one liter per day. Strauss advises the following diet in enteroptotic individuals:

Breakfast: Flour soup with butter, gruels, cocoa with milk and cream mixture, eggs and buttered rolls.

Dinner: Dishes made of flour, eggs and cream.

Afternoon: Crackers with milk and cream mixture, zwieback and butter.

Supper: Rich flour soups or dishes made from flour and eggs, with tea and milk-cream mixture.

Bedtime: Milk-cream mixture.

Diet in Nervous Affections of the Intestines.—In nervous affections of the intestines the most indigestible food is well borne, while digestible food causes discomfort, and it is therefore best to regulate the diet according to the patient's digestive power. At times these patients are in a much enfeebled state, and a liberal diet is therefore indicated. Frequently a well regulated rest cure is needed in order to restore the patient to health. When an excessive amount of gas accumulates in the intestine, due to nervous conditions, such food as cider, fruit and cabbage should be avoided.

Diet in Chronic Diarrhea.—The diet in chronic diarrhea must vary with the type of the disorder. In the nervous variety, the patient must be instructed to restrain the bowel movements, except early in the morning. Food which has a tendency to increase the bowel movements must be avoided; especially to be noted are foods containing much cellulose, pickles, salads, cabbage, turnips, fruit and carbonated waters. Among the foods to be recommended are tea, broth, cooked cereals, boiled milk and gruels; at times milk even when boiled disagrees and causes diarrhea. In a certain group of cases, however, excellent results have been obtained in the treatment of chronic diarrhea by means of an exclusive milk diet. In other cases systematic rest cures are required in order to bring about favorable results. The author has utilized the following diet list for moderate cases of chronic diarrhea:

Eight o'clock, A. M.:	
200 grams of cocoa (cooked in water)	45 calories
2 soft-boiled eggs	160 calories
50 grams of zwieback	179 calories
Ten o'clock, A. M.:	
250 grams of broth with 1 egg	80 calories
Twelve o'clock, noon:	
250 grams of broiled chicken	212 calories
200 grams of mashed potatoes	245 calories
50 grams of zwieback	179 calories
Four o'clock, P. M.:	
1 soft-boiled egg	80 calories
200 grams of cocoa (cooked in water)	45 calories
50 grams of zwieback	179 calories
Seven o'clock, P. M.:	
100 grams of rice cooked in bouillon	34 calories
200 grams of sweetbread	180 calories
50 grams of wheat bread	130 calories
Nine o'clock, P. M.:	
100 grams of raw scraped beef	118 calories
50 grams of zwieback	179 calories
Total	2045 calories

Diet in Chronic Constipation.—It is necessary in prescribing a diet in chronic constipation, that the food consumed should be of such a nature as to increase intestinal peristalsis. Such foods contain a large percentage of cellulose and therefore by producing a large residue in the bowel are especially useful for this condition. If a large glass of cold water, to which a pinch of salt has been added, is taken before breakfast, it is often useful in aiding the bowel movements. Raw and stewed fruits are especially beneficial when taken on an empty stomach morning and evening. It is most important in all instances that the patient be required to realize the necessity of having bowel movements at the same hour each day. The smoking of a cigar in the morning will frequently stimulate peristaltic movements. Constipation is caused often by dyspeptic affections and is frequently relieved by the treatment directed to the gastric disorder. The use of drugs should as far as possible be avoided and proper exercise should be indulged in, in addition to the dietetic treatment. Of the foods that are useful in the treatment of chronic constipation are cabbage, asparagus, peas, cauliflower, onions, spinach, tomatoes, celery, salads, oatmeal, whole wheat, corn, Graham, rye, and bran bread, as well as honey, molasses, cider and raw fruits. Dates, figs, and prunes are especially useful. It is always important to prescribe a sufficient quantity of water in chronic constipation inasmuch as this condition is frequently due to insufficient water. Constipating foods must be avoided as tea, chocolate, cocoa, port wine, rice, barley and farina gruels. Milk acts in some instances as a laxative; in others it produces diarrhea; it must therefore be tested in every instance; buttermilk, however, usually has a laxative effect. By careful regulation of the diet alone, many cases of chronic constipation have been entirely cured. The following diet list taken from Friedenwald and Ruhräh's "Diet in Health and Disease" has been found useful by the author in the treatment of patients affected with chronic constipation:

Six o'clock, A. M.:	
40 grams of orange juice	88 calories
o'clock, A. M.:	
200 grams of milk with coffee	192 calories
2 soft-boiled eggs	160 calories
150 grams of Graham bread	375 calories
60 grams of butter	326 calories
Ten o'clock, A. M.:	
400 grams of cider	280 calories
Twelve o'clock, noon:	
200 grams of broth, with 1 egg	84 calories
100 grams of steak	214 calories
100 grams of carrots	41 calories
100 grams of beans	193 calories
150 grams of Graham bread	375 calories
200 grams of stewed apples	106 calories
Four o'clock, P. M.:	
400 grams of buttermilk	166 calories
Seven o'clock, P. M.:	
100 grams of scraped beef	118 calories
150 grams of Graham bread	375 calories
200 grams of stewed prunes	176 calories
300 grams of cider	210 calories
Nine o'clock, P. M.:	
40 grams of figs (or 400 grams of buttermilk)	46 calories
Total	3525 calories

Diet in Chronic Intestinal Stasis.—Intestinal stasis is frequently associated with chronic constipation. This condition is due frequently to certain bands and membranes, often adhesions developing on the peritoneum from the bowel, and producing kinks. Due to this condition, intestinal toxemia may develop, which is responsible for many of the general and nervous manifestations. The diet to be prescribed for this condition is much like that described under the head of chronic constipation. Water must be taken liberally, and vegetables, fruits, and salads given in abundance. In many instances a radical change should be made in the diet in order to alter the bacterial flora. The diet must be of such a nature as to leave considerable residue in the bowel.

Diet in Hemorrhoidal Affections.—Inasmuch as chronic constipation is associated with this affection it is important that this condition be overcome. Patients having hemorrhoids should eat moderately and should avoid all excesses of food and drink. Moderate exercise should be indulged in and the bowels should be evacuated daily. Patients affected with hemorrhoids must avoid spiced foods, cheese, beans, cabbage and fruits\containing seeds. They may take carrots, spinach, potatoes, and asparagus and stewed and raw fruits; meats, eggs, and fish are to be recommended.

DIET IN DISEASES OF THE LIVER

Biliousness is a term frequently applied to a class of symptoms associated with disturbances of the liver, and often accompanied with constipation. In the acute attacks it is necessary to restrict the diet for some days, following the administration of calomel. The object of treatment in all liver conditions is to give this organ as limited amount of work as possible and the food should therefore be easily digested, and consist in general of a mixture of proteins, fats and carbohydrates. It may, however, be necessary in those instances in which the liver function is markedly impaired, to avoid the use of fats and carbohydrates entirely for some days.

In all liver affections overeating is apt to produce annoying symptoms and the abuse of alcohol may cause serious changes in the liver; it is therefore important to avoid the use of alcohol. The foods producing an irritating effect on the liver are spices, mustard, pickles, peppers, concentrated meat broths, radishes and horseradish; these as well as strong coffee and tea should be forbidden.

In serious liver disturbances, the diet should be limited to diluted milk, gruels, buttermilk and fruit juices. In milder forms the diet may be increased, including lean meat, curd, junket, bread, toast, zwieback, fresh fruit and green vegetables; mineral waters may be taken if dropsy is not present and act best when taken on rising and between meals. At times hot water is an excellent remedy in these affections. Food must be well masticated and taken in small quantities and best at frequent intervals. Whenever there is a tendency to dropsy the Karell diet is indicated together with a restricted salt intake.

Diet in Catarrhal Jaundice.—In the early period of this affection, when the patient is nauseated and is suffering with indigestion, he must be kept quiet in bed and upon a liquid diet. Milk is the most suitable form of food in this condition and should be diluted with lime water or Vichy. Kumiss and buttermilk are often well borne and may be allowed as well as broth (oysters, clam, chicken) and albumen. Food is best prescribed in small amounts, given at two-hour intervals. As soon as the stomach is less irritable zwieback, toast, chicken, sweetbread, and chops may be allowed, and later thickened flour soup, mashed and baked potatoes, white fish, green vegetables, stewed fruit and fruit juices may be added to the dietary. All indigestible food must be avoided for a considerable period of time after the jaundice has disappeared. Coffee and tea, which may be allowed after the acute period of this affection, should always be weak; alcoholic stimulants must be avoided, and fats, butter and cream, as well as an excess of starch and sugar, should be abstained from for a considerable period of time. Mineral waters are of considerable service during the entire course of the disease; of these Carlsbad, Vichy, Saratoga and Bedford waters are most beneficial.

Diet in Cholelithiasis.—In the dietetic treatment of gall-stone disease the meals should be so arranged as not to be too widely separated. The patient must be advised against prolonged fasting as the ingestion of food increases the biliary flow, while fasting has a tendency to cause the bile to be retained in the gall-bladder. Kehr considers it advisable to eat late at night, while others believe it most desirable to awaken the patient in the middle of the night to administer food. Naunyn advises a large breakfast and a large meal should follow a period of fasting. According to the author's experience, patients affected with this disease are apt to err in the direction of consuming more food than can be thoroughly digested and in consequence have indigestion, which may be the immediate cause of an attack of colic. In this condition, certain foods must be avoided; fats should be reduced much in quantity and given only in the most easily digestible form. Herter has demonstrated that animals fed for months largely on a fat diet and a low protein diet are apt to have concretions in the gall-bladder. Excesses of starches and sugar must also be avoided as well as all rich foods. Of meats only the lean varieties should be utilized. Milk and eggs are permissible and green vegetables and fresh fruit may be allowed. Cereals may be taken moderately and potatoes may be given in small amounts; the heavier vegetables, as turnips, beets, and cabbage, must be forbidden. Bread should be eaten stale or in the form of toast or zwieback. Alcohol must be forbidden but light coffee or tea may be allowed. Certain mineral waters as Carlsbad, Vichy, Bedford and Saratoga are useful; the best effects are obtained, however, when the water is taken at the spring itself.

Rothschild and Rosenthal have shown that in the majority of cases of cholelithiasis a hypercholesterinemia is present and that inasmuch as the cholesterin content of the blood is dependent upon the cholesterin content of the food, by diminishing the latter one can correspondingly diminish the former. This is accomplished by placing the patient upon a fat-free diet which excludes lipoids and renders difficult the esterification of the free cholesterin of the food.

All foods rich in fats are excluded and butter, olive oil, cocoa, eggs, cream, meat, and fish must be avoided. Only vegetables, cereals together with skimmed milk, and sugar may be allowed. By means of these dietetic restrictions Rothschild and Rosenthal have been able to reduce the hypercholesterinemia which may be an etiologic factor in chronic cholelithiasis.

Diet in Cirrhosis.—As soon as the diagnosis is established, if ascites is not present, the patient should be placed on a milk diet or at least a diet of milk foods. Buttermilk and kumiss are especially to be recommended and eggs, albumen and cereals added to milk are valuable. The saline mineral waters are of great service and should be taken on arising. As the patient improves, the easily digestible meats as chicken, lamb chops, the green vegetables, baked potato, fruit, eggs, stale bread and toast are to be recommended. When ascites exists, a restricted salt-free, dry diet is of value. Hot water may be allowed in small amounts at intervals during the day but not more than 800 to 1000 c.c. of fluids should be allowed per day. A Karell diet is frequently indicated. Alcoholic beverages are to be forbidden.

DIET IN DISEASES OF THE PANCREAS

When the diagnosis of pancreatic disease has been definitely established, fats and protein foods should ordinarily be greatly restricted. Cream, oil and butter should be allowed only in exceedingly moderate amounts and meats and eggs should also be permitted only in small quantities. Milk is allowable in moderate amounts while smoked beef should be prohibited.

The carbohydrates may ordinarily be allowed in adequate quantities, providing, however, that the symptoms of diabetes are not at hand. If diabetes is present, an antidiabetic diet must be prescribed. In order to assist the digestion of fat as well as the proteins, pancreatin should be prescribed after meals while small quantities of hydrochloric acid may be administered before meals to increase the pancreatic secretion.

DIET IN PERITONITIS

In acute peritonitis the diet is of importance only until operation is to be performed. The patient should abstain from all food and nutrient enemata resorted to if necessary. The stomach must be washed out frequently and continuous enteroclysis is practiced according to the Murphy method; if the patient no longer vomits and the operation has not been performed, fluids may be given in the form of milk with lime water, bouillon and egg albumen. In order to stimulate the patient small quantities of sherry, whiskey, brandy or champagne may be utilized.

In chronic peritonitis, the diet should consist of food given in the most digestible form, such as boiled meats, eggs, milk, cereals, and vegetables in the purée form; food should be taken in moderate quantities at frequent intervals.

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I

CHAPTER VIII

DISEASES OF THE TONSILS

By M. L. Breitstein, A.B., M.D.

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GENERAL DISCUSSION

Introduction.—It is highly significant that in a practice of medicine a chapter should be given to special consideration of diseases of the tonsils. For while diseases of the tonsils were recognized and treated by Hippocrates, it is only recently that the relationship between tonsilar affections and systematic disease has been recognized. In this chapter the author will not attempt to consider affections of the mucous membrane of the tonsils, but only those diseases which are peculiar to the tonsils as lymphoid tissue. There is no attempt to consider surgical procedures.

Definition.—Adenoid tissue widely distributed in the upper air passages consists of a delicate reticulum of connective tissue filled with lymph cells or leukocytes. The leukocytes may be collected into small clusters called follicles. These follicles in turn may be grouped into larger masses which are then called tonsils. The aggregation of tonsils in the region of the pharynx are united by tracts of mucosa, containing well-marked infiltrations of lymphoid tissue, and they have been called by Waldeyer[1] the lymphatic pharyngeal ring. This ring is composed of the following parts:

(1) The pharyngeal tonsil (adenoids, tonsil of Luschka, and the third tonsil). This is a single mass of lymphoid tissue found in the roof of the naso-pharynx.

(2) The palatine tonsils (faucial tonsils). These are generally referred to as *the tonsils.* There are two palatine tonsils and one is to be found in each of the recesses between the palatine arches. These arches are referred to generally as the anterior and posterior pillars respectively. The anterior pillar contains the palato-glossus muscle, and the posterior pillar contains the palato-pharyngeus muscle.

(3) The lingual tonsils. There are two lingual tonsils situated one on each side of the midline, at the base of the tongue, and lying in front of the valleculæ.

It is to be noted that the findings of lymphoid tissue extending in a diffuse and irregular manner downward into the larynx or upwards in scattered masses into the naso-pharynx is not to be regarded as an abnormal finding. The distribution of the pharyngeal lymphoid tissue has wide individual variations. It is a mistake to view the individual divisions of the pharyngeal lymphoid ring as separate and distinct, and to ascribe to them independent pathological significance. They are to be regarded rather in their entirety as the lymphatic apparatus of the pharynx, just as all of the solitary follicles and Peyer's patches of the intestine represent the lymphatic system of that organ.

Anatomical Considerations.—EMBRYOLOGY.—The lingual and palatine tonsils of Waldeyer's ring are formed from the mesoblastic area of the second arch and the cleft behind it. The lingual tonsil is formed about the thyro-glossal diverticulum. The pharyngeal tonsil (adenoids) is a mesoblastic thickening in the roof of the pharynx around the recessus medius which is a depression—perhaps connected with the notochord—placed a little distance behind the junction of the stomodæum and fore-gut.[2, 3, 4]

HISTOLOGY.—Tonsils, as we have noted above, are composed of lymphatic cells collected into groups, called follicles, which are separated from one another by areas of connective tissue. The surface of the tonsil is generally irregular, being indented with crypts (Lacunæ or sulci). The epithelial covering varies with the situation. Thus the palatine and lingual tonsils are covered with flat epithelium; the pharyngeal tonsil, under normal conditions, is covered with ciliated epithelium. The palatine tonsil is partly enclosed in a connective tissue capsule; that portion of the tonsil which is seen in the oral cavity is free of capsule and the extent of this exposed area has individual variations. From this connective tissue capsule there project septa of variable thicknesses, and they extend to a point just under the epithelium. The tonsil tissue is not compact, but between the connective tissue septa the parenchyma forms folds and recesses. These recesses vary in number and in depth. In general the pharyngeal tonsil has the same structure as the palatine tonsil.

Blood Supply.—The tonsil receives its blood supply from:

(1) The tonsillar and ascending palatine branches of the facial artery. These enter the tonsil in its lower half.

(2) The descending palatine (branch) of internal maxillary artery sends a small branch to the superior lobe.

(3) The dorsalis linguæ artery supplies the anterior pillar and gives branches to the lower half of the tonsil.

The veins drain into the tonsillar plexus on the lateral side of the tonsil, which is an extension of the pharyngeal venous plexus.

Lymph Drainage.—It is important to consider the lymph drainage of the tonsil. *The tonsils differ from the other lymphatic glands in that, as yet, there has been no anatomical demonstration of afferent lymphatics to the tonsil.* The efferent lymphatics of the tonsil pass through the fibrous trabeculæ and the capsule, pierce the superior constrictor muscle of the pharynx and empty into the tonsillar gland (Wood).

The tonsillar gland is one of the superior deep cervical glands lying under the anterior border of the sterno-cleido-mastoid muscle, in the angle between the internal jugular and facial veins. It is, perhaps, the most constant in position of all the cervical glands. When enlarged it projects anteriorly beyond the anterior border of the sterno-cleido-mastoid muscle and appears superficially about ½ to ¾ inches below the angle of the jaw.

The deep cervical glands are divided into a superior and inferior group. The superior glands, 10 to 16 in number, extend along the course of the internal jugular vein from the tip of the mastoid to the level at which the vein is crossed by the omohyoid muscle. Clinically, these glands are divided into an anterior and posterior group by the sterno-cleido-mastoid muscle.

The posterior glands receive afferent vessels from (1) the superficial glands in the suboccipital region, (2) the retro-pharyngeal glands, and (3) the mucosa of the pharynx and naso-pharynx and the nasal mucosa posteriorly, including that of the sinuses. When these glands are enlarged it is difficult to feel them as discrete nodes; they form rather an indefinite swelling under the posterior border of the sterno-cleido-mastoid.

It can be seen from the preceding that an enlargement of these posterior glands without an involvement of the anterior glands cannot be attributed to the tonsils.

The anterior glands form a chain lying just under the anterior border of the sterno-cleido-mastoid. They receive their afferent vessels from the tonsils, the base of the tongue and from the submaxillary and submental nodes. Vessels from the body of the tongue also go to one of the anterior glands.

If the tonsillar gland enlarges alone and there is no lesion at the base of the tongue, the enlargement may be attributed to infection through the tonsil. If the submaxillary glands are enlarged at the same time the source of the infection should be looked for anteriorly, *i. e.*, lesions of the gums, tongue, cheeks or an abscessed tooth.

In the majority of those cases in which both the posterior and anterior cervical glands are involved, the infection is probably of double origin; the lateral and posterior pharyngeal nodules or the adenoid tissue of the vault being responsible for the posterior enlargement.

The inferior group of the cervical glands receive afferents directly from the upper nodes. They are of importance in connection with the tonsils chiefly because of the possibility of tubercular infection reaching the pulmonary apices via this route (Wood[5], Grober)[6]. In view of the frequency with which primary tuberculosis of the tonsils occurs, the existence of this possible line of invasion between them and the lungs assumes clinical importance. It should be said, however, that this mode of pulmonary infection is probably most infrequent.

Function of the Tonsils.—We have no certain knowledge of the function of the tonsils. It is highly improbable, however, that the tonsils are vestigeal in nature and without function. The histological structure is essentially the same as lymph glands in other parts of the respiratory and alimentary tracts and it is probable that their physiological significance is the same. Among the theories which have been advanced for the function of the tonsil may be mentioned:

(1) *The Hemopoietic Theory.*—This theory ascribes to tonsils the production of lymphocytes and rests upon definite histological findings.

(2) *Internal Secretion Theory.*—This theory advanced by Masini[7] has no clinical evidence to support it.

(3) *The Protective Theory.*—This theory attributes to the tonsil a protective power against bacterial invasion. The question must ever be one of the mastery between the two forces contending; on the one hand between the virulence and number of the organisms in the crypts, and on the other, the forces of resistance of the individual whether they be local and inherent in the tonsil, or general depending on anti-bacterial conditions of the blood and lymph. It is evident that a tonsil already laden with infection is no longer a protection but a positive danger.

(4) *The Eliminating Theory.*—Ashhurst[8] asserts that the tonsils act as eliminating organs during the common acute infections. Such secondary involvement as an acute tonsilitis following a septic lesion of the hand and arm is so interpreted by him. This manner of involvement does take place during the course of septic infection in other parts of the body. This might be explained by the great susceptibility of the tonsils and other lymphoid nodules of the pharynx to infections which have entered the blood stream.

(5) *The Immunity Theory* (Good[9]).—This theory assumes that in the tonsil organisms and toxins are generated in just sufficient quantities as will produce the necessary anti-bodies to immunize the individual against the active invasion of the organisms themselves. The tonsils of

the child would be of great importance until the immunity to the prevalent bacteria of his environment had been acquired.

(6) *Fleischmann's Theory.*—Recently Fleischmann[10] advanced the theory, based upon experiment, that the physiological role of the adenoid tissue is a secretion of an easily oxidized substance. It satisfies, in this manner, the oxidation requirements of the acids of the mouth and nasal cavity and protects the tissue from the harmful effects of the acid. This has been refuted by Amersbach, Koenigsfield and others.[11]

General Considerations of the Pathology and Bacteriology of the Tonsils.—Conditions in the tonsil would appear to be ideal for the invasion of bacteria and the absorption of the toxic products of their growth. The tonsil crypts are tortuous and deep and their mouths are very frequently closed. The result is that a certain amount of débris is found in the crypts of all tonsils. The cryptic epithelium is thin, showing only one or two layers of cells and often it is very difficult to demonstrate microscopically. So there is very little resistance to the entrance into the parenchyma of the tonsil of foreign particles from the crypts (experiments of Goodale, Hendlesohn and others have verified this). These conditions do not hold at birth but are the results of development attained at about the end of the second year. Bacteria appear in the tonsillar crypts at about the time of the bacterial invasion of the intestinal canal (from twenty-four to seventy-two hours after birth), and plasma cells appear in the lymphoid tissue shortly after—according to Davis[12] during the second or third week of life. As they are usually present in chronic inflammatory lesions, they are probably in the tonsils an expression of chronic irritation due to absorption of bacterial toxins from the crypts. The so-called "buried tonsil" is much more liable to marked cryptic retention than is the prominent, pedunculated tonsil. Acute inflammatory attacks and hypertrophy are important factors in increasing the difficulty of drainage from the crypts; the first by creating cicatricial tissue and the second by increasing the depths of the crypts and the amount of cellular material which passes into them from the lymphoid tissue. The débris of the crypts is largely cellular-lymphocytes, polynuclear leukocytes and exfoliated epithelium — containing also cholesterin crystals, hyalin material, and large numbers of bacteria. It is of a cheesy consistency, white, yellow or green in color and often has an offensive odor. Fein[13] says that this débris is normally pressed out of the tonsil by the muscular action in swallowing. When for any reason swallowing is painful, as in acute inflammation of the tonsils, the débris accumulates more readily and this explains the "white spots" on the tonsils. The "white spots" can be seen in dysphagia due to diseases of the tongue, the larynx or the esophagus, even though the tonsils are quite healthy. Often it is impossible to tell the condition of the deeper part of the crypt until the tonsils have been removed. Only then can retention material and abscesses be seen close to the capsule. Latent primary tubercular foci are found only after the tonsils have been removed. Of a total of 1000 tonsillectomies reported by Crowe (*loc. cit.*), 46 were found to be tuberculous. Barnes[14] reports 3 tuberculous foci out of 150 tonsils removed from patients with no evidence of tuberculosis elsewhere.

BACTERIOLOGY.—Many varieties of organisms have been isolated from the tonsillar crypts; of these the streptococcus is the most frequently present. Davis[15] reports the examination of 113 tonsils removed from patients with systemic lesions suspected to be of tonsillar origin or in whom there had been frequent attacks of tonsillitis.

The predominating organisms were:

Hemolytic streptococci in 90 cases.
Pneumococcus in 8 cases.
Staphylococcus albus in 6 cases.
Influenza bacillus in 3 cases.
Diphtheria bacillus in 2 cases.
Bacillus mucosus capsulatus in 1 case.

Dwyer and Gignoux[16] report bacteriological findings from essentially normal tonsils *in situ*. The variety of organisms is greater than in the report of Davis. The Klebs-Loeffler bacillus was found in 7 per cent. of the cases and the staphylococcus in 28 per cent. Among others the bacillus pyocyaneous and Friedlander's bacillus are reported. More recently Bloomfield[17] reports that of 85 unselected normal individuals, 5.8 per cent. were found to be carriers of Friedlander's bacillus and *that the breeding place of the bacteria is in the tonsil.*

After examining repeated cultures from the throats of the same healthy individuals over a considerable length of time Bloomfield[18] concludes that the organisms fall into two groups: (*a*) The true normal flora including non-hemolytic streptococci and Gram-negative cocci, and (*b*) pathogenic and non-pathogenic organisms which are accidently introduced and are present usually only a short time in a given individual.

The combined statistics of a large number of investigators show that primary tuberculosis of the tonsils is found in 6 per cent. of all cases.[19] Organisms resembling the fungus of actinomycosis have been reported in the detritus of the crypts.[20]

DISEASES OF THE PHARYNGEAL TONSIL, OR ADENOIDS

The most frequent abnormality seen is hypertrophy of the adenoids. Why hypertrophy of adenoids should occur in some individuals and not in others is a problem yet to be solved. It is seen most frequently in children between 3 and 12 years of age; cases as early as the third month have been reported. Adenoids have a tendency to "run in families." The condition is somewhat more common in males than in females. Nasal obstruction in children favors the growth of the adenoids. It is claimed that this tendency to adenoids is not so marked in warm dry climates as in cold, damp and changeable ones. The lymphatic tissue often markedly increases in size during and after acute infectious diseases, such as measles, scarlet fever, diphtheria and whooping-cough. Adenoids are very commonly associated with enlarged tonsils.

Symptomatology.—The symptoms of enlarged adenoids are for the most part the indirect result of nasopharyngeal obstruction. The most common of these are the following:

(1) Stasis of nasal secretion with excoriation of the skin about the external nares.

(2) Accessory nasal sinus infection.

(3) "Adenoid facies." Retardation of mental and physical development.

(4) Defective speech.

(5) Affections of the ear, both catarrhal and suppurative.

(6) Reflex neuroses. Among these the most common are: night terrors, asthma, petit-mal-like attacks, paroxysmal nocturnal attacks of coughing, enuresis nocturna.

(7) Spontaneous bleeding from the nasopharynx in children is not an infrequent symptom of enlarged adenoids.

(8) Chronic mucopurulent catarrh. Hypertrophic rhinitis.

(9) Frequent coryza.

A further result of enlarged adenoids is an interference with the development of the superior maxillary bones, which results in a high, narrow, V-shaped deformity of the hard palate.

Diagnosis.—A diagnosis of nasopharyngeal obstruction is easily made from the symptoms. Direct examination of the nasopharynx in children is difficult; in older children posterior rhinoscopy will show the adenoids. In younger children the digital examination of the nasopharynx will confirm the diagnosis made from the history.

DIFFERENTIAL DIAGNOSIS.—The other conditions to be considered are:

(1) Polypus.
(2) Fibroid tumor.
(3) Malignant growths.

The immobility of a polypus and the presence of polypi in the nasal cavity, differentiates the polypus from the adenoids. Fibroid tumor is much harder and is freely movable. Malignant neoplasms are of rapid growth and are accompanied by pain, spontaneous hemorrhage, fetid discharge and progressive emaciation. These conditions are rare. The pathological nature of these neoplasms will be considered under the general head of neoplasms of tonsils.

Treatment.—The treatment for enlarged adenoids is **surgical removal**, the remarkable beneficial effects of which is now universally recognized.*

Pathology.—The upper and posterior wall of the nasopharynx is normally the seat of a mass of lymphoid tissue. The pathological considerations for hypertrophy in addition to those above noted are the same as those for the palatine tonsils. These will be considered later.

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*Adenoidectomy was first done and advocated by Hans Wilhelm Meyer in 1868.

DISEASES OF THE PALATINE TONSIL

The mucous membrane of the palatine tonsils may share in any of the inflammatory affections which attack the pharynx. They are commonly inflamed in association with the lingual and pharyngeal tonsils (Waldeyer's ring) in infectious catarrhs of the upper air tract. The more specific inflammations of the tonsils have been classified as follows:

(1) Catarrhal tonsillitis or superficial tonsillar angina.

(2) Follicular tonsillitis (also called lacunar, croupous, or cryptic tonsillitis).

(3) Parenchymatous tonsillitis.

(4) Suppurative tonsillitis (tonsillar—peritonsillar).

As the first three affections differ chiefly according to the degree and extent of the inflammation and as they often shade into one another, we shall consider them together.

Etiology.—Tonsillitis is undoubtedly an infectious disease, which is associated with various micro-organisms and may be induced by a number of causes, as follows:

(*a*) Tonsillitis is most frequently met with between the ages of ten and thirty. It sometimes occurs in children, and it is rare after fifty years of age.

(*b*) The condition of the tonsils forms an important predisposing factor, as inflammation more readily occurs in glands that are enlarged and have been the seat of previous inflammation or calcareous deposits.

(*c*) It occurs as the initial stage of measles and scarlatina (also in convalescence of scarlatina and diphtheria) and is frequently present in diphtheria and in secondary syphilis.

(*d*) Water and milk may carry the infection.[21]

(*e*) The disease is rarest in summer and most frequent in spring and autumn.

(*f*) Infection from the nose and nasopharynx (sinus disease) may cause recurring tonsillitis.

Symptoms and Clinical Course.—It is not necessary that the symptoms of tonsillitis be referable to the throat. Not uncommonly a patient may complain of malaise, anorexia, thirst, prostration and some fever (temperature 100° to 103° F. [37.2° to 39.4° C.]) and it is only on examination of the throat that an explanation is found in a condition of subacute follicular tonsillitis.

In the majority of cases local symptoms make themselves felt at once. A patient then complains of headache, backache, pains in the limbs, general malaise and at the same time he has a sense of fulness in the throat, or sharp pain, which radiates toward the ear and is greatly augmented on attempts to swallow. As the isthmus of the fauces becomes encroached upon, the voice becomes muffled and thick. The senses of smell and taste are suspended. *Not infrequently there is some deafness from the Eustachian catarrh—but the spread of actual inflammation to the middle ear is relatively infrequent.* Both tonsils are usually affected, the glands and the angle of the jaw are swollen and in most cases very tender. The temperature is always raised varying from 100° to 103° F. (37.2° to 39.4° C.); often there are recurring remissions and these must not be viewed too favorably in the absence of any rebate of the local symptoms.

In the milder forms of catarrhal and follicular tonsillitis the process may only last three or four days, but it is apt to go on increasing until the fifth day, declining at the seventh with an abatement of the general symptoms, often leaving some slight dysphagia for some time.

Examination.—The breath is usually fetid. The tongue is coated and in acute cases is protruded with difficulty. The tonsils are seen to be larger than usual and reddened. The reddening involves the anterior pillars, the uvula, the soft palate and the pharynx. The congested surface of the tonsil is dotted with irregular islets of debris which occlude the orifices of the crypts. This material can be wiped away, but it tends to form again. The pharyngeal tonsil (adenoids) frequently shows the same follicular inflammation. Now and then these collections are not limited to the mouths of the crypts but extend over the intervening tonsil surface in the form of a false membrane and *most rarely,* on to the soft palate and uvula. *This false membrane is white, gray or dirty yellow in color, is easily separated and leaves an abraded but not bleeding surface beneath.*

When the parenchyma of the gland is more involved, the local symptoms are much more marked. The whole region is festooned with loops of thick tenacious mucus which the patient is only able to clear away with painful difficulty. There is always a more or less marked pharyngitis. Constipation is a frequent part of the disease. The urine is scanty, high colored and charged with urates. It seldom contains any albumin, and, when it does it usually appears as a late phenomenon and, therefore, as a complication of the disease. *This is a distinguishing point from diphtheria.*

It is said that the spleen is often enlarged.[22]

Diagnosis.—This generally presents no difficulty. In peritonsillitis the tonsil itself is often slightly inflamed, or not at all. A redness or smooth bulging may be observed on one side of the soft palate. It is pertinent, at this point, to emphasize the points which distinguish follicular tonsillitis from diphtheria. Diphtheria is more gradual in its onset, and is more apt to be limited to one side, or at least, it does not start on one side and then develop on the other, as is generally the case in tonsillitis. There is less pain and fever with diphtheria, the urine is much more frequently albuminous in the early stages of the disease. In diphtheria the membrane is seldom limited to the tonsils but invades the uvula and soft palate. The spread of the membrane in follicular tonsillitis is uncommon and in no case does it, as in diphtheria, spread to the larynx, nasopharynx or nose. In both affections the cervical glands are enlarged. The younger the child, the more apt is the disease to be diphtheria. The positive diagnosis is to be made by a culture for the Klebs-Loeffler bacillus.

Complications.—Reference has already been made to the possible supervention, particularly in children, of specific infectious diseases. In all stages a watch should be kept for the development of any joint or cardiac lesions. The urine should be examined for possible albuminuria. Any weakness of the palate or any local paralyses would indicate that the case had been one of diphtheria which had been overlooked. The ear drums should be frequently inspected. Among the unusual complications of tonsillitis which have been reported are: orchitis, oöphoritis, pleuropneumonia, acute osteomyelitis, pyemia and septic manifestations. Angina pectoris has been recorded as a sequella of tonsillitis.

Treatment.—The treatment of tonsillitis is based on the recognition that there is a general infection. The patient should be **kept in bed and isolated,** as the disease is infectious. **Nourishment should be liquid.** The constitutional treatment is best begun with a brisk purge. As there is difficulty in swallowing pills, 2 to 4 grains (.13 to .26 gram) **calomel** with 3 grains (.195 gram) of **soda bicarbonate** can be placed on the tongue and washed down with a sip of milk. It is good to follow this in eight hours with a few teaspoonfuls of **Rochelle salts. Salicylates, aspirin** and **phenacetin** are indicated. There is a variance of opinion as to the efficacy of the use of **gargles** and the local application of antiseptics. Syringing or irrigation of the pharynx is more efficacious than gargling. A mild antiseptic can be used. A favorite prescription is one teaspoonful each of salt, soda and borax to a quart of boiling water. The solutions should be used as hot as they can comfortably be borne. Local applications of **silver nitrate, argyrol, guaiacol** or **mercurochrome** are used.

Prognosis.—In simple tonsillitis the prognosis is usually favorable. The disease runs its course usually in a week. It may drag along two or three weeks, however. Care should be exercised not to overlook the earlier manifestations of the more virulent septic forms which may result in edematous, erysipelatous, or phlegmonous laryngitis or in purulent cervical cellulitis (angina Ludowici), spreading to the mediastinal glands; or in general infection with resulting endocarditis or pericarditis, infective phlebitis, peritonitis, orchitis or ovaritis.

Pathology.—If the disease is limited to the catarrhal variety, the surface of the tonsil shows the changes common to inflammation of the mucous membrane. In the follicular variety the changes are extended to the crypts which become filled with epithelial cells, fibrin, leukocytes and bacteria. These form an amorphous mass which fills the crypts and appears at their mouths as discrete patches of white, gray, or yellow exudation. In the parenchymatous form there is marked leukocytosis into the lymphatic nodules, and congestion of the whole gland. Small discrete, intrafollicular foci of suppuration occur.

SUPPURATIVE TONSILLITIS (QUINSY, SORE THROAT)

Suppurative tonsillitis is most frequently seen in the form of suppurative peritonsillitis or quinsy. A peritonsillar abscess is the result of suppuration behind the tonsillar capsule. The pus generally extends upwards into the soft palate, often just beyond the supra-tonsillar fossa, into which it ultimately breaks. It may extend forward toward the anterior or backward into the posterior pillar; in the latter case sometimes coming to the surface on the lateral pharyngeal wall. It occasionally breaks through the anterior wall of the soft palate and in front of the anterior pillar.

In spite of the extent and position of a peritonsillar abscess the pus is, with very rare exceptions, limited to the palate and tonsillar region. Extension of pus into the deep structure of the neck occasionally occurs, but considering the frequency of the disease this complication is very rare.

Etiology.—The causes for peritonsillar abscesses are practically the same as those for acute tonsillitis. It occurs most frequently at the ages from eighteen to thirty-five. In appearance a tonsil from which an abscess is liable to start does not necessarily differ from any other. It often belongs to the submerged type, in which the openings of the crypts are hidden and obstructed.

Symptomatology.—At the onset the symptoms of peritonsillar abscess are much like those of acute tonsillitis. The pain gradually increases on the affected side. The temperature varies and increases with the formation of pus. On inspection the soft palate is swollen and the uvula is pushed away from the affected side. As the abscess increases, the tonsil is pushed toward the median line, and the soft palate on the affected side becomes bulging and reddened. The duration of the abscess before spontaneous rupture varies, but it is most often about a week. After free drainage of the pus the symptoms rapidly disappear. The prognosis is good. Edema of the larynx may occur in that variety in which pus burrowing downward along the lateral wall of the pharynx occurs.

Treatment.—During the first few days it is better to wait until pus has formed, using **gargles** of various alkaline solutions. Whenever there is a marked bulging of the soft palate immediately above the tonsil it is presumptive evidence that pus has already formed external and superior to the tonsil. Various methods of **opening the abscess** are suggested. An incision directly into the bulging supratonsillar fossa is most frequently practiced. Recently tonsillectomy has been suggested for the treatment of peritonsillar abscess. This is a procedure of doubtful value and not without danger. The general treatment for peritonsillar abscess is the same as that for acute tonsillitis.

CHRONIC TONSILLITIS AND CHRONIC ENLARGEMENT OF THE TONSILS

Synonyms.—Hypertrophic tonsillitis; hyperplastic tonsillitis; chronic lacunar (or follicular) tonsillitis; chronic parenchymatous hyperplasia; chronic fibroid degeneration of tonsils.

Enlarged tonsils are not necessarily pathological. In children there are always lymphoid glands between the pillars of the fauces. If they are large in infancy they tend to atrophy at puberty. This, of course, is not a rule without exceptions. In some instances the hypertrophy first appears towards puberty. Many enlargements of the tonsils are purely inflammatory and temporary and not true hypertrophies.

Etiology.—The causes of lymphoid hypertrophy are not definitely known. Enlarged tonsils "run in families" and the tendency to hypertrophy seems to be heredity. Sex seems to have no influence. Hypertrophy has been attributed to mouth breathing, but this is very unlikely. Scarlatina, measles, diphtheria, whooping-cough, influenza, small-pox, and repeated attacks of tonsillitis predispose to hypertrophy of the tonsils. Hypertrophy of the tonsils in children is practically always associated with hypertrophy of the adenoids in the nasopharynx. This is not true in adults. Frequency of hypertrophied tonsils in school children varies from 33.4 per cent. to 39.48 per cent. according to St. Clair Thomson.[23]

Symptomatology.—The tone of the voice is frequently thick and mealy. The enlargement of the tonsils is so gradual that unless they are inflamed the patients are seldom conscious of their presence. Some may complain as of the feeling of a "lump in the throat." Eustachian catarrh may be kept up by the interference with the action of the levator palati and salpingo-pharyngeus muscles. The cervical glands are apt to be chronically enlarged. Chronic or recurrent follicular tonsillitis is frequent and peritonsillitis may be associated with a small amount of tonsillar hypertrophy. The patient sometimes complains of bad breath and of being able to expectorate masses of bad tasting, cheesy material. Cough is sometimes attributable to the presence of large diseased tonsils. The symptoms of nasal obstruction are frequently due to the associated enlargement of the adenoids.

Diagnosis.—This rarely presents any difficulty. The enlargement is practically always symmetrical, and unilateral enlargement should always be looked upon with suspicion, particularly in adults. Malignancy of the tonsil may develop so insidiously as to be mistaken for a simple hypertrophy.

Treatment.—This may be divided into: (1) Local; (2) general; (3) surgical; and (4) x-ray. The local and general treatment are the same as those used for acute tonsillitis. Surgical and x-ray treatment will be considered below.

Pathology.—There is an increase of the normal structure of the tonsil. Three principal varieties may be considered: (1) Chronic follicular type, in which the crypts are more marked and contain a variable amount of mucus and altered epithelium undergoing fatty degeneration; (2) chronic parenchymatous hyperplasia, in which the overgrowth of lymphoid tissue is the chief change; and (3) chronic fibroid degeneration, in which there is a marked increase in the connective tissue stroma. This type, by compression, causes atrophy of the lymph nodules and obstruction of the crypts. Occlusion of these latter cause the formation of cysts and the deposit of calcareous masses.

In most instances as a result of frequent inflammatory attacks the tonsils are adherent to one or both pillars.

NEOPLASMS OF THE TONSIL

Neoplasms of the tonsils are uncommon. They may be benign or malignant. The benign tumors seldom or never ulcerate and they grow very slowly. Malignant neoplasms, on the other hand, usually ulcerate and grow more rapidly.

Benign Neoplasms.—These include papilloma, fibroma, lipoma, angioma, and fibro-enchondroma. The papilloma and fibroma are the least uncommon. Lipoma, angioma and fibro-enchondroma are very rare findings. Papilloma springs from the surface mucosa and consists of branching fibrous stalks surrounded by thickened epithelium. The multiplication of the fibrous stalks produces numerous cauliflower projections springing from a common base. The growths are therefore commonly pedunculated. They may be single or multiple. Whenever such a growth occurs in a person over forty years of age, malignancy should be suspected and piece of tissue removed for microscopic examination.

Fibroma of the tonsil is a hard, rounded, smooth tumor, usually single, and may be sessile or pedunculated. It is much less vascular than the naso-pharyngeal fibroma. It occurs during adolescence or young adult life. Lipoma and enchondroma do not differ from fibroma in gross appearance.

Microscopical examination is necessary for diagnosis.

Angioma can be recognized by its purple, nodular surface, and its soft, almost fluctuating consistency.

TREATMENT.—The **papillomata** should be **removed.** This is most simply done by **tonsillectomy.** Angioma presents some difficulty because of the danger of hemorrhage. **Cauterizing** procedures therefore are preferable. If the growth is well limited to the tonsil, tonsillectomy is the best treatment. Other benign neoplasms need not be removed unless their size causes discomfort or obstruction. In this event tonsillectomy is indicated.

Malignant Neoplasms.—Carcinoma and sarcoma of the tonsils occur. Carcinoma usually occurs in later life. It rarely occurs before the fortieth year and in the majority of cases after the fiftieth year. Sarcoma has a predilection for the young and middle aged. It may occur at any time of life, however. Carcinoma occurs more frequently in males than in females. It may be primary in the tonsils; usually, however, it is secondary; invading the tonsil by direct extension from the base of the tongue or from the faucial pillars. These growths tend to ulcerate and growth is usually rapid; though frequently in older individuals, the growth may be very slow. Cervical glands usually enlarge early. Carcinomata may be confused with syphilitic ulcerations. The history, the therapeutic test, the Wassermann reaction and finally the microscopic tissue examination serve to differentiate the two. The symptoms of carcinoma are ulceration, pain which may be severe and lancinating in character, often shooting to the ear on the affected side, loss of weight, cachexia, and early involvement of the cervical glands. The last symptom, together with the age of the patient, usually suggest the correct diagnosis.

Sarcoma of the tonsil arises from the cells of its fibrous supporting framework and may be of the spindle or round cell type. The lymphosarcoma, if it is primary in the tonsil, probably arises from the lymphoblasts of the germinal centers of the lymph follicles. Lymphosarcoma may be secondary to such growth in the lymphatic glands of the neck (Hodgkin's Disease). It occasionally is involved in the general course of a lymphatic leukemia. Clinically the sarcoma may simulate the fibroma if the growth is slow.

TREATMENT.—The treatment of malignant tumors of the tonsils is through **extirpation,** *if there is little glandular enlargement and no extensive infiltration into the surrounding tissue.* In advanced stages palliative treatment alone is warranted. **Tracheotomy** and **gastrotomy** may be indicated. **Radium** is much more effective in sarcoma than in carcinoma. **Diathermy** is of value as palliative treatment.

PROGNOSIS.—The prognosis in carcinoma is very grave. The progress may appear slow before ulceration occurs, but then it advances rapidly, so that a case may terminate fatally in six to eighteen months. The outlook in sarcoma is more hopeful, as it remains localized for a longer time and its progress is slow. Death may occur from cachexia, inanition, hemorrhage, suffocation, or intercurrent disease.

SEPTIC SORE THROAT

Septic sore throat constitutes such an important chapter in medical history that it deserves separate consideration.

Including the Baltimore epidemic up to the year 1912 there were reported 22 epidemics of septic sore throat, all of which were milkborne.[21] In practically all, the symptoms were the same. The streptococcus was the reported bacteriological findings whenever such examination was made.* "The malady was characterized by a sudden onset, high and irregular fever, inflammation of the fauces of varying degree, marked enlargement of the cervical glands, particularly in the case of children, and a course much longer and attended with many more complications than the usual type of tonsillitis."

The appearance of the throat varied. Every type of faucial inflammation was represented. The most frequent lesion was a dusky red discoloration of the fauces, a faucitis. Among other findings may be mentioned: follicular tonsillitis, a membranous exudate resembling that of diphtheria, peritonsillitis with abscess formation. In most severe cases the constitutional disturbances were much more severe than the faucial symptoms. Absence of the tonsils did not preclude the development of the infection. Hamburger divides the cases into three groups, as follows:

(1) Those characterized by inflammation of the fauces associated with marked visible enlargement of the cervical glands.

(2) Those with or without the enlargement of the cervical glands, but presenting some metastatic septic or streptococcic complication, such as erysipelas, arthritis, abscesses or peritonitis.

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* In the Baltimore epidemic the streptococcus was of the hemolytic type.

(3) Cases of sore throat occurring in households in which one or both of the first two types prevailed.

Treatment.—The treatment of sufferers of septic sore throat did not vary from the usual management of febrile patients. No advantage seemed to be derived from direct faucial medication. Aspirin was used. Hexamethylenamin was used but without apparent benefit. Vaccine was not employed. Altogether 600 cases were reported in the Baltimore epidemic, though there were probably a great many more. There were a total of 31 deaths of which 26 were almost certainly due to this infection. Of the 16 deaths recorded by Hamburger, 9 were due to peritonitis, 4 were due to sepsis and 1 was due to erysipelas.

TONSILLOTOMY AND TONSILLECTOMY

Tonsillotomy was done by Celsus. He speaks of this method as follows: *"hamulo excipere et scapello excidere."*

The older idea of the surgical treatment of the tonsils by tonsillotomy has been generally abandoned for tonsillectomy or complete enucleation. There is no evidence that complete enucleation of the tonsils has any disadvantageous results. The occasional scarring of the palate and fauces is absent or trifling in skilled hands.

Tonsillotomy is the incomplete removal of a diseased gland and the post-operative scar tissue formation by more effectively blocking the crypts does great harm. Whenever surgical treatment of the tonsil is indicated, tonsillectomy is the operation of choice. As tonsillectomy sometimes produces a functional effect in the singing voice, the only instance in which tonsillotomy is to be considered is in cases of professional singer in whom the only symptom is obstruction to respiration.

Indications for Tonsillectomy.—The indications for tonsillectomy may be grouped as follows:

(1) Local disorders in the upper air passages:
  (*a*) Hyperplasia of the tonsils causing difficulty in swallowing, articulation or breathing.
  (*b*) Frequent tonsillitis or quinsy.
  (*c*) Chronic laryngitis or bronchitis may often be helped.
  (*d*) Chronic catarrh or suppurative otitis media or an eustachian tube affection.
  (*e*) Chronic diphtheria carriers. Some of these cases will clear up after the removal of the tonsils and adenoids.
  (*f*) New growths.
(2) For local trouble in the cervical glands draining the tonsils:
  (*a*) Simple hyperplasia of the glands at the angle of the jaw.
  (*b*) Tuberculous cervical adenitis.
(3) For general systemic disorders secondary to a focus of infection in the tonsils.
  (*a*) Infectious arthritis, in which the peri-articular changes predominate.

  - (*b*) Myalgia or myositis.
  - (*c*) Early stages of a glomerulo-nephritis.
  - (*d*) The various nervous symptoms designated by "neurasthenia," eneuresis nocturna, and sometimes convulsive seizures resembling petit-mal.
  - (*e*) Occasionally an iritis (if it is not luetic or tuberculous) is benefitted by tonsillectomy.
  - (*f*) Rare conditions in which the chief symptoms are septic temperature, leukocytosis, general muscular and joint pains; general intoxication with negative findings on physical examination except for large succulent tonsils.
- (4) As a prophylactic measure.
  - (*a*) In chorea, acute rheumatic fever and heart lesions it is often advisable to remove the tonsils and adenoids in order to prevent further cardiac lesions that may result from an acute tonsillitis.
  - (*b*) In cases of chronic nephritis and arteriosclerosis that give a history of repeated attacks of tonsillitis.

Contra-indications for Tonsillectomy.—(1) Tonsillectomy should never be undertaken during the acute stage of tonsillitis. It is best to wait at least three weeks after all symptoms have subsided.

(2) A tonsillectomy is of no benefit in the chronic deforming types of arthritis.

(3) There is nothing to be gained from a tonsillectomy during the acute stage of chorea, acute rheumatic fever or endocarditis.

(4) Tonsillectomy should not be done in children under ten years of age, unless the indications are marked.

(5) All conditions wherein a general or local anesthetic is dangerous, are to be classed as contra-indications to tonsillectomy, such as pulmonary tuberculosis, status lymphaticus, diabetes, etc.

(6) Hemophilia. The possibility of this condition should be carefully investigated.

Postoperative Complications of Tonsillectomy.—The following are some of the postoperative complications of tonsillectomy and their frequency as reported by Crowe[24] in 1,000 cases:

Complications	*Frequency*
Postoperative pneumonia	2
Acute otitis media	4
Suppurative cervical adenitis	2
Tetany	1
Erysipelas	1

In addition lung abscess as a complication occurs, particularly when the bleeding at the time of the operation is not carefully controlled. In one New York hospital, 8 cases of lung abscess following tonsillectomy were observed in one year.[25]

X-RAY AND RADIUM IN THE THERAPY OF DISEASES OF THE TONSILS

At this time the reported experience with x-ray and radium in the treatment of tonsils does not warrant the substitution of such treatment for tonsillectomy.[26]

The fact that lympathic tissue is more easily destroyed by x-ray than any other living tissue furnishes the basis for x-ray treatment of the tonsils. Murphy, Witherbee, Craig, Hussey, and Sturm of the Rockefeller Institute, made a joint study of 46 cases of diseased tonsils treated with x-ray and found the following: "They could bring about a definite diminution of the size of hypertrophied tonsils, and that with such atrophy there was evidence of less occluded débris in the crypts, with a diminution in the exudate and sepsis, particularly in reference to hemolytic streptococci, and without producing undue reaction either constitutionally or locally."

These authors do not infer that the x-ray producing the lymphoid atrophy has any bactericidal action.[27, 28] The bactericidal effect of beta radiation on suspension of organisms,[29] and upon apical teeth abscesses[30] has been demonstrated. Simpson states that he is positive he can cause the complete atrophy and disappearance of any but a *fibrous tonsil* in from one to three treatments.[31] More experience is necessary before the value of this method is known in effecting the disappearance of systemic involvements of tonsillar origin.

TONSILS AND FOCAL INFECTION

As this subject has been exhaustively considered in this work by Dr. Joseph S. Evans (Vol. I, p. 303), the author shall quote only from some follow-up observation[32] so that we may be able to judge the type of lesion in which improvement may be expected after the removal of infected tonsils and adenoids.

Arthritis Group.—These cases are divided into four general heads as follows:

INFECTIOUS ARTHRITIS.—The predominating change is in the periarticular tissue often with an effusion into the joint cavity. The onset is usually insidious or may come on two or three weeks after an acute tonsillitis. Only one joint may be involved but in the majority of cases many of the joints are affected. There is very little, if any, elevation of temperature and generally no redness around the affected joints. The joints are swollen and often extremely painful on motion. There is no associated endocarditis. Tonsils and adenoids were removed in 91 such cases. Thirty-one cases were followed; of these, 24 showed normal joints at the last examination. Often it was from six to eight months before all the joint symptoms cleared up.

MYALGIA.—There was stiffness, tenderness, pain on motion, and sometimes a continuous throbbing pain. Four such cases were followed after tonsillectomy. Two cases were examined one year after operation and two, two years after operation. All four cases had entirely cleared up.

One must be careful in these cases not to form a *post hoc propter hoc* judgment.

RHEUMATOID ARTHRITIS.—Patients in this group are for the most part middle aged people. This disease is usually well advanced when they seek medical advice. Nine such cases were followed. Two of the cases were improved but five became decidedly worse after tonsillectomy. The conclusion drawn from this group is that only in very exceptional circumstances should the tonsils be removed in a case of rheumatoid arthritis.

ACUTE RHEUMATIC FEVER.—Four of 25 cases had a recurrence after leaving the hospital. One patient was well for two years after removal of his tonsils and then had a characteristic attack of rheumatic fever.

CHOREA.—From follow up observations in 24 cases, Crowe concludes that removal of the tonsils and adenoids is not a very satisfactory therapeutic or prophylactic measure in chorea.

Nephritis.—Tonsillectomy is of value in that form known as glomerulonephritis described by Baehr.[83] In these cases there is a comparatively recent history of tonsillitis, arthritis, acute rheumatic fever, or some other form of a general streptococcus infection with or without endocarditis. Of 18 cases in which tonsillectomy was done, 13 cases showed a normal renal condition, from ten months to three years after operation.

Hyperplasia of the Cervical Glands.—A chronic enlargement of the deep cervical glands at the angle of the jaw, together with the past history of the patient, was the indication for tonsillectomy in 541 of the 1,000 cases reported. The majority of these cases were from seven to ten years of age; 366 cases were followed at periods varying from six months to four years after tonsillectomy.

The experience showed that the tonsils are the most common site of chronic infections that give rise to a hyperplasia of the deep cervical glands near the angle of the jaw. In 122 of the 366 cases no glands were palpable at examination subsequent to the operation and in 187 they were just palpable at the angle of the jaw.

Tonsillectomy alone will not cure a tuberculous cervical adenitis, an arthritis or a glomerular nephritis. It is necessary in these cases to carry out all general measures that will tend to increase the patient's resistance. If the tonsils are the primary focus of infection, however, their removal may naturally alter the prognosis by preventing a constant reinfection.

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CHAPTER IX

ESOPHAGOSCOPY

By Waitman F. Zinn, M.D., F.A.C.S.

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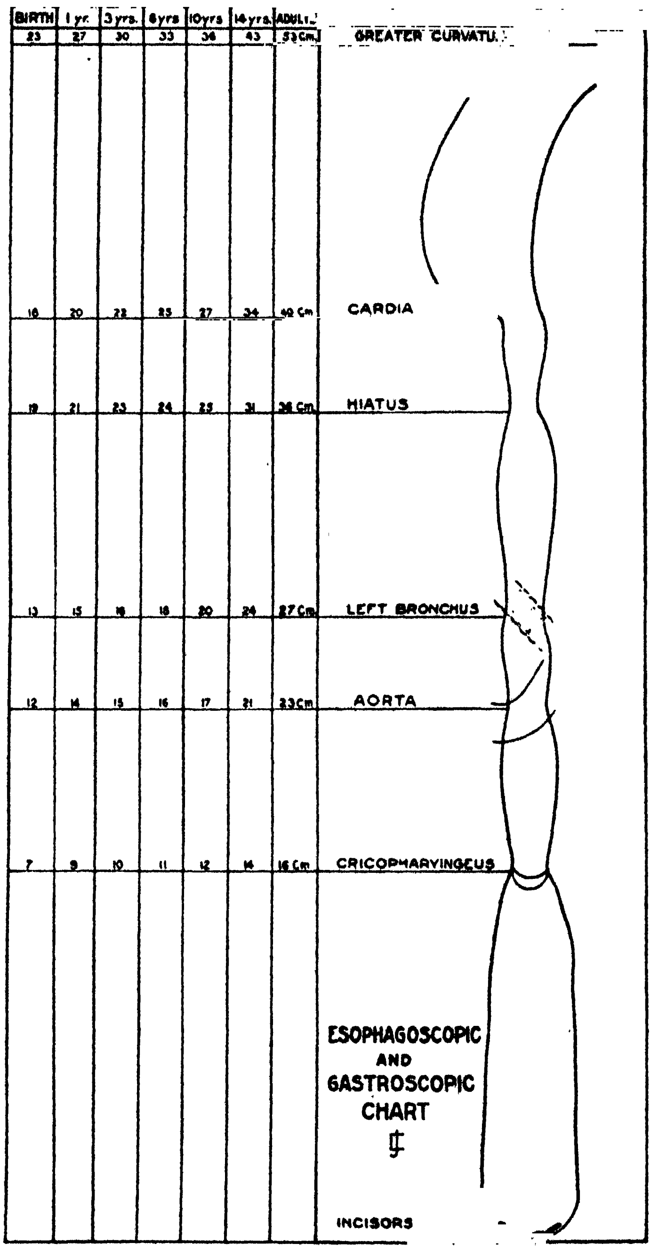
ESOPHAGOSCOPY

The art and knowledge of endoscopy has developed during the past decade to such a degree that at the present time in the hands of the specialist it is practised with but little danger. Esophagoscopy is performed for the purpose of diagnosing and treating diseased conditions and removing foreign bodies from the esophagus.

Instruments and Technic.—The instruments and technic have been perfected so that it is no longer difficult to acquire skill in the passage of the esophagoscope. The teaching facilities at present have been brought to a high level. Formerly, the passing of bougies was the only known means of examining the diseased esophagus. The procedure was practised, even though it was generally recognized as very dangerous. With the advent of the direct, ocular inspection of the lumen of the esophagus, the passing of bougies by tactile sense alone is no longer justified. The esophagoscope affords a direct, ocular vision, gives ample opportunity to examine the condition of the esophageal walls and permits the use of tactile sense, transmitted either by the instrument passed through the scope or by means of the end of the tip, in noting the firmness of the walls and the encroachment of any growth from without upon the esophagus. Specimens for pathological examination may be removed from the lesion with specially designed forceps.

The success one attains and the ease with which the instrument is passed depend greatly upon the position of the patient when it is introduced. The esophagus is not a straight tube, but assumes the course of the cervical and dorsal segments of the spinal cord, extending slightly to the left in the normal subject. This position varies in pathological cases. The author has employed the position known as the "Chevalier Jackson high-low position." This affords ample opportunity to inspect the walls and locate any small lesion. Often foreign bodies may be passed unnoticed if the lumen is not kept continually in view. The esophageal secretions, which are usually watery, may be removed through the aspirating tube to which a hand suction pump is attached by means of a rubber tube. When the number of cases justifies it, a motor-driven



BIRTH	1 yr.	3 yrs.	6 yrs	10 yrs	14 yrs.	ADULT.	
23	27	30	33	36	43	53 Cm.	GREATER CURVATU
18	20	22	25	27	34	40 Cm	CARDIA
17	21	23	24	25	31	36 Cm.	HIATUS
13	15	16	18	20	24	27 Cm.	LEFT BRONCHUS
12	14	15	16	17	21	23 Cm	AORTA
7	9	10	11	12	14	16 Cm	CRICOPHARYNGEUS
							INCISORS

FIG. I.

Chevalier Jackson's esophagoscopic chart of approximate distances of the esophageal narrowings from the upper incisors prepared from measurements in the living. Arranged for convenient reference during esophagoscopy in the dorsally recumbent patient.

suction pump may be utilized instead of the hand suction pump, provided the amount of suction is regulated.

The beginner should never attempt an esophagoscopy upon the living without first passing the scope on the cadaver and thoroughly familiarizing himself with the direction, anatomical narrowings and general appearance of the normal esophagus. He should select as his first case, when possible, an adult, and where there is absence of the upper teeth, he will find the operation much less difficult. Skill should be acquired both in tactile sense and in the art of handling the instruments.

Contra-indications.—Water hunger is probably the most important and frequent contra-indication. In such cases, it is customary to postpone the esophagoscopy until the vitality of the patient has been partly restored by rectal feeding or through gastrostomy. In acute inflammatory conditions following the ingestion of caustic poisonings, the passage of the tube serves no purpose and should be avoided. In patients with large aneurysms, the procedure is dangerous, but with beginning aneurysm, where there is little danger of rupture, a diagnosis of the condition may be made with little risk.

Indications.—According to Jackson, any case showing an abnormal sensation referable to the region or function of the esophagus calls for an esophagoscopy. Diseases of the esophagus cannot be excluded without direct ocular inspection. Bougies frequently pass by foreign bodies without giving the slightest indication of their presence. They may push the foreign body farther down and in the case of sharp pointed objects may cause them to penetrate the wall of the organ. The removal of foreign bodies by means of "coin catchers" and other blind methods has resulted so often in the death of the patient that their use should be entirely prohibited. Since the development of esophagoscopy diseases of the esophagus have been more accurately diagnosed and curable lesions may receive treatment in time to effect a cure.

Dangers and Complications.—The dangers and complications of esophagoscopy are few in the hands of the trained. When an attempt is made to pass the instrument without direct vision of the lumen or where undue pressure is exerted on passing it too rapidly, perforation of the walls of the esophagus may result. Injury to the right arytenoid, gouging of the esophageal wall, resulting in sloughing and perforation, may follow. The symptoms usually following esophagoscopy are slight stiffness of the neck, pain in the back, which lasts for a few hours only.

Anesthesia.—The esophagus is not a sensitive organ, except at the entrance from the pharynx, and according to Jackson, neither a local nor a general anesthesia is necessary in adults or children. On the other hand, Mosher prefers a general anesthesia in most cases. In the author's experience, no anesthesia is needed except in rare instances. A hypodermic injection of morphin prior to the operation helps to secure the necessary relaxation, but under no circumstances should the examination be proceeded with upon a struggling and resisting patient.

Foreign Bodies.—The symptoms produced by the lodgment of a foreign body in the esophagus vary according to the size, shape and contour of the object, those that are small or smooth producing the least pronounced symptoms. The points at which a foreign body is most likely to be arrested in its passage through the esophagus are determined by the normal anatomical constrictions. These are the entrance of the esophagus, the point of crossing of the aorta, the crossing of the left bronchus, and the cardia. The distance of these points from the upper incisor teeth at various ages are given in the above chart abstracted from Jackson's "Peroral Endoscopy."

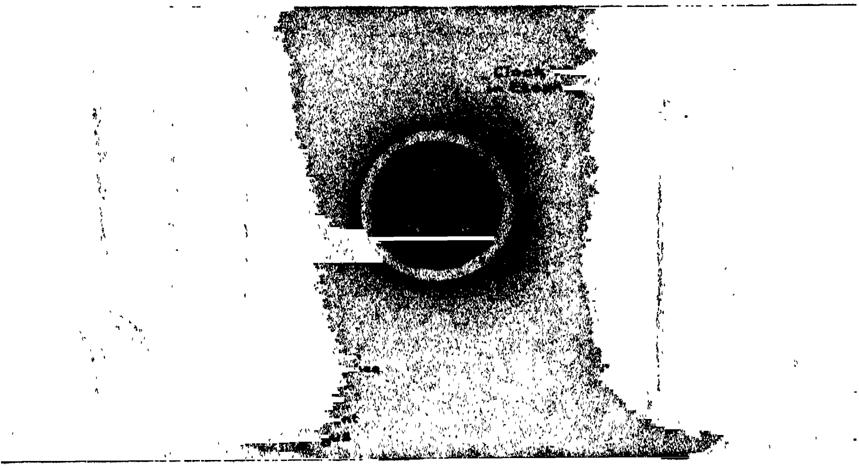


Fig. 11 (*a*)
Illustration showing piece of beef bone 27 cm. from central incisors at bifurcation of trachea in the esophagus of man, aged 25. Patient was referred to author on fourth day after lodgment of foreign body. Removal by esophagoscopy. No anesthesia.

Fig. 11 (*b*)
Illustration showing foreign body 23 cm. from central incisors at left bronchial crossing in esophagus of patient one and a half years old. This was removed without anesthetic. No after-results.

By far the safest, quickest and most satisfactory method of removing a foreign body from the esophagus is direct esophagoscopy. The elaborate and complicated armamentarium for this purpose has been brought to a high state of perfection, especially in the United States, and the refined technic has been developed by esophagoscopists in all parts of the country. The details of technic are matters which are of particular concern to the practical specialist, and space does not permit their consideration. Some of the problems which confront the esophagoscopist, however, are of interest.

The simplest of all cases is a small coin lodged just below the cricoid of a young child, and promptly brought to esophagoscopy. Without anesthesia of any kind, an esophagoscope or a speculum specially made for the purpose is introduced and the upper edge of the coin comes easily into view. After making certain that the edge is free and not embedded in swollen or ulcerated mucous membrane, it is seized with forceps, and under gentle but steady traction, the coin is brought out together with the tube. There is no bleeding and no trauma and, as the entire procedure is completed in a fraction of a minute, there is no shock. No after-treatment is needed.

A more difficult, though by no means infrequent situation, is an open safety pin lodged in the thoracic esophagus in the neighborhood of the large vessels. Various instruments have been devised for freeing the point and closing the pin and many such objects have been successfully removed.

Each foreign body presents problems of its own; some require careful study and the first attempt is not always successful. With care, skill and patience, practically every foreign body which has entered the esophagus through the mouth, can be removed through the natural passage.

Cancer.—Cancer of the esophagus is not an infrequent disease. According to Willy Meyer it is fifth in frequency of all places in which cancer occurs. It is the least malignant of all cancers affecting the gastro-intestinal tract. The early symptoms may exist for a long time before the diagnosis is made.

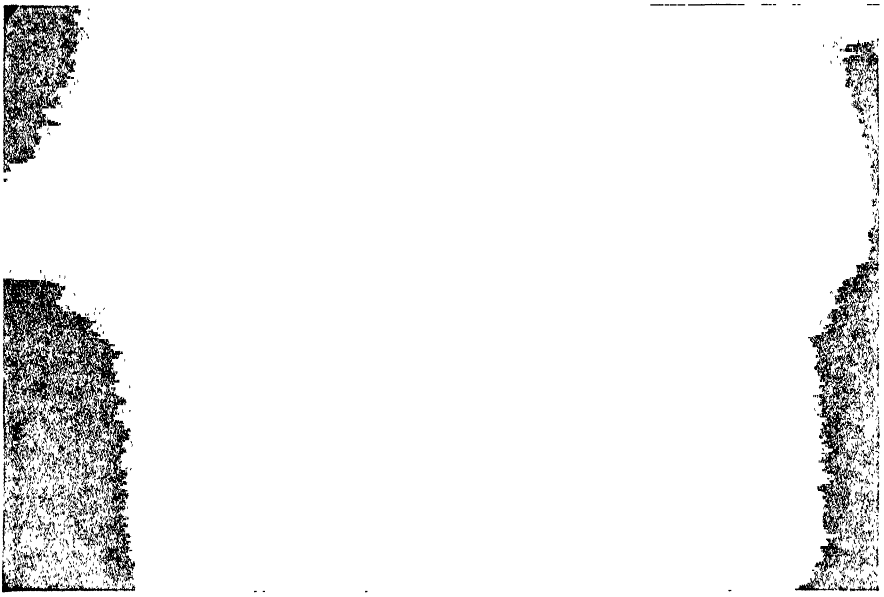


Fig. III.

Radiograph of foreign body (twenty-five cent piece) in the esophagus of a child three and a half years old. Removed by esophagoscopy without anesthesia. (Radiograph by Dr. Wetherbee F. Fort.)

Formerly, the treatment of cancer of the esophagus was entirely symptomatic with gastrostomy performed in the later stages. It is true that the percentage of recoveries is small, but this is due largely to the fact that nearly all the cases reach the hands of the surgeon late in the disease, when the patient's vitality is reduced and when adhesions of the esophagus to the surrounding organs, penetration of the cancerous growth into the mediastinum and enlargement of the mediastinal glands increase the difficulties of the operation. Furthermore, the cases that have remained well after the operation are cases that were fortunately operated in the early stages of the disease. It is therefore imperative that the diagnosis should be made at a much earlier period in the course of the disease than is at present customary. *The function of esophagoscopy in cancer of the esophagus is to arrive at an early diagnosis.*

In advanced cases, the diagnosis can be made by inspection. The large, granular masses, the many-lobed cauliflower excrescences can hardly be mistaken. As about 90 per cent. of all growths in the esophagus are carcinomatous, the diagnosis of the presence of a growth almost suffices. In early cases, the diagnosis is not so easily made. The new growth may resemble a leukoplakia, or a syphilitic or tuberculous ulceration. Simple ulcerations and areas of thickened mucosa, due to aberrant patches of columnar ciliated epithelium, such as are occasionally found low down in the esophagus, must also be differentiated. These finer differences cannot be recognized by the naked eye, and necessitate the removal of specimens for microscopic examination. Some surgeons object to this procedure, because the irritation of the small wound stimulates the growth of the tumor, but in the early stages the diagnosis cannot be made without it. Esophagoscopy aided by biopsy, can establish the diagnosis beyond any question as soon as the tumor makes its very first appearance.

However, in order that the cases should be recognized at a time when they are still amenable to surgical intervention, the author wishes to emphasize the fact that it is necessary for the profession generally to remodel completely the mental picture of the disease which has been inherited from ancient times. The middle-aged man, in apparently good health, who complains of slight gastro-intestinal disturbance, who finds that food occasionally has a tendency to remain in the esophagus for a short time, and upon whom an esophagoscopy reveals the presence of a small tumor, is the patient upon whom the surgeon can operate with some hope of success. Nervous dyspepsia and globus hystericus are common errors in diagnosis. No diagnosis of gastro-intestinal symptoms referable to the esophagus can be considered as complete unless malignancy of the esophagus has been excluded.

In the treatment of cancer of the esophagus, esophagoscopy plays only a limited rôle. According to Mosher, the cauliflower excrescences may be excised and the lumen of the esophagus temporarily restored. Radium treatment can be carried out directly through the esophagoscope, either by inserting radium needles into the growth, or by injecting radium "seeds." The accurate emplacement of the radium seeds presents some difficulties.

Compression Stenosis of the Esophagus.—Compression or "kinking" of the esophagus may be caused by a tumor originating in any of the organs surrounding the esophagus. The degree of compression is not usually extreme in these cases, so that the symptoms of which the patient complains are generally referable to the tumor itself, and not to the esophagus. Occasionally, however, the dysphagia is noticed by the patient, and sometimes, a large bolus of food becomes arrested above the constriction, causing a sudden and complete obstruction. Among the causes of such obstruction may be mentioned enlarged tubercular glands, aneurysm, enlarged left auricle, goiter, and mediastinal tumors, both malignant and benign.

Esophagoscopy must be conducted with extreme caution in these cases, particular care being taken to avoid passing beyond the obstruction. This is especially true in aneurysm, although the author has made the diagnosis of aneurysm of the aortic arch at a time when the aneurysm was too small to be recognized by radiography; the diagnosis was confirmed at a much later date.

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CHAPTER X

DISEASES OF THE ESOPHAGUS

By Harlow Brooks, M.D.

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MALFORMATIONS

Congenital malformations of the esophagus are very rare, except in monsters. They are but very infrequently seen in viable infants and it is extremely infrequent that the condition becomes of any great clinical importance. The most common malformation is a failure to fuse of the pouch representing the developing pharynx and the bud directed upward from the foregut. As Prudden has shown such lesions are almost invariably associated with other congenital stigmata and these infants rarely survive sufficiently long to justify anything in the way of corrective surgery, which, of course, offers the only possibility of relief. Gastric fistula with subsequent corrective operation has been suggested, but insofar as the author has been able to learn it has never been successfully performed. The infant usually dies from inanition, from an aspiration pneumonia, or from associated defective conditions. The possibility of diverticula of congenital origin has been suggested by most authors, and it has been admitted even by Zenker and Ziemmsen. From the nature of the evolution of the esophagus, however, it is apparent that it must be an extremely unusual occurrence and its significance is of academic interest only. Occasionally the esophagus is divided longitudinally by a septum which converts it into a two-chambered tube. This lesion need not necessarily be fatal. A not infrequent malformation is for a fistulous opening to exist between the trachea, or large bronchi, and the esophagus. If such an infant be viable, death takes place soon from aspiration pneumonia. F. Göppert[3] has recently reported an exceedingly interesting instance of this character. Congenital stricture of the esophagus, naturally most probable at the point of juncture of the pharangeal pouch and the foregut, is a possible condition but certainly very rare. From its nature either early death takes place from inanition or it tends to correct itself.

ESOPHAGITIS

Esophagitis may be conveniently considered under the usual headings of acute and chronic, depending upon the activity or chronicity of the condition, but chronic forms frequently develop from acute causes, and acute changes may appear at any time in chronic as well as in acute inflammatory conditions.

Etiology.—Acute inflammatory disease of the esophagus may result from the swallowing of any of the irritants, acids, alkalies, mustard water, carbolic acid, sulphuric acid, and so on indefinitely. Hot fluids as tea, hot water, hot soups as well as a large group of toxic substances may also cause its appearance. It may appear in any of the acute exanthemata, chicken-pox, small-pox and particularly in scarlet fever, measles and German measles. It may follow as an extension downward of any of the forms of stomatitis, as in diphtheria or thrush. It may occur in practically all of the chronic inflammatory diseases, as in syphilis, tuberculosis or in the subacute types of infection as in typhoid fever and the like. It has been reported in rheumatism. Esophagitis either of the acute or chronic form may follow by extension inflammatory diseases of adjacent structures as mediastinal abscess, high Pott's disease, gummata and so on. It may develop from impacted or perforating foreign bodies, from splinters of bone, bits of metal, or from dry, hard materials of any kind. It may result from fermentation of normal food products in diverticula or in stricture. It may develop during the administration of certain drugs, as mercury, the iodids, and perhaps with the salicylates and bromids.

Symptomatology.—Naturally the severity and to a considerable degree the character of the symptoms of esophagitis depend in a very large part upon the character and gravity of the lesions or disease causing the inflammation. It is a common finding of the deadhouse to discover entirely unsuspected and oftentimes extensive lesions of the esophagus of which no complaints whatever had been made. The esophagus is not a particularly sensitive tissue or viscus. Were it otherwise, many of us would have to materially reform our habits and tastes of eating, but it is quite surprising oftentimes to find the degree to which serious lesions may develop without the production of striking symptoms. Pain and discomfort on swallowing are probably the most frequent indication. They may be so severe that the patient refuses foods entirely. The fluids are as a rule more readily taken than solids, but sometimes the opposite is the case. No actual pain may be present but simply a discomfort which, being located back of the sternum, is frequently accounted for by the patient on the basis of cardiac disease. Physicians also probably make the same error more frequently than most of us appreciate. The pain or discomfort, while almost invariably excited by the act of deglutition, may persist for a long time after, or, particularly in the case of abscess, it may even be continuous and only vaguely associated with the taking of food. In many cases, especially in the acute ones, the discomfort may be so severe that the patient refuses to swallow the saliva—an act which is commonly automatically carried out—, and it may then dribble out of the mouth. Fever may or may not be present, depending upon the character and degree of the lesion. In abscess, phlegmonous esophagitis and, after extensive destructive processes, a septic course of temperature is ordinarily present together with a polynuclear leukocytosis in case actual suppuration is present. Asthenia and a degree of prostration out of proportion to that dependent merely upon lack of nourishment will develop in the serious cases. Pain and tenderness along the course of the tube is present generally and the head and neck will be held rigidly, so much so that a meningeal irritation may be suspected on casual examination. Other patients complain only of a dull, oppressive pain in the precordium, usually, but not always, intensified on swallowing. Where perforation or abscess formation has taken place, displacement of the trachea or larynx with corresponding tracheitis, dyspnea, or laryngitis may appear. Abscess may be present anywhere in the neck or lower down in the mediastinum.

Diagnosis.—As a rule diagnosis is not particularly difficult, although detection of the basic causative condition may involve considerable study. More or less secretion is discharged into the buccal cavity and from its character some opinion of the process may be formed. In simple catarrhal processes the secretion is largely mixed with saliva but contains mucus, usually many leukocytes and a few red blood-cells. Pus will occur in the phlegmonous processes and where necrosis of the membrane is taking place particles of tissue may be discharged which in case of corrosive poisoning may have an absolutely diagnostic appearance. The symptom of pain and discomfort on swallowing which will usually be found associated with a suggestive history of onset and probable etiology will usually suffice for a tentative diagnosis while physical examination with the careful use of the bougie or stomach tube will usually render diagnosis positive. In chronic cases more difficulty is generally experienced and here the use of the esophagoscope is of the greatest possible assistance. The character of the material which collects in the mouth should be studied, and, taken in association with the general symptoms and history, it will usually permit of a sufficiently accurate diagnosis as to the type of the process present to properly direct treatment. Where a membranous process is present, bacterial cultures are, of course, indicated and in all available instances microscopic and chemical study of the exudate, if it be ejected into the mouth, should be made.

Treatment.—In most acute cases the primary requisite is **feeding by rectum, or through the duodenal tube** until such time as swallowing is possible without undue suffering on the part of the patient. When the duodenal tube can be passed without too much difficulty it is by all means the most desirable procedure since it may then be left in place and the patient may be satisfactorily nourished for an indefinite period of time. Rectal feeding is in most cases very unsatisfactory because it cannot be continued long enough to give the desired rest to the esophagus without seriously compromising the nourishment of the patient, thereby greatly reducing his resistance. In some instances the **Rehfuss tube** may be passed if necessary after cocainization of the channel, but standard size stomach tubes should not be used unless the physician is reasonably certain that they may be employed without danger of perforation. In acute corrosive esophagitis alkalies or mild acids may be employed to wash out the esophagus as indicated by the nature of corrosive poison taken. **Lime water** and **magnesia** may be safely used in any instance, however, and they are also extremely beneficial in many types of chronic esophagitis. As a rule warm, bland demulcent drinks may be best employed while swallowing is possible without too great suffering, but in some cases iced drinks are better tolerated. Where abscess formation is present it should be treated **surgically** as soon as possible, and in occasional cases of erosive esophagitis direct application through the esophagoscope is indicated, but this instrument must be used with extreme care in cases of this kind because of the possible infliction of serious damage with the instrument. After the acute phases of the inflammation have passed over, it is well to pass the bougie occasionally in order that stricture formation may be lessened or obviated. In any instance the treatment of esophagitis demands highly individual methods, determined in most part by the etiology or by the character of the lesions present. The main object must be to give rest to the organ for as long a period as practical, to favor healing processes and to prevent stricture.

Prognosis.—The prognosis in esophagitis is extremely indefinite until the exact nature, extent and etiology of the lesion present are fully determined. It is very uncertain, especially in the corrosive type of the disease, both as to the present outcome, and as to future complications.

Pathological Anatomy.—The pathological anatomy and the form or type of the inflammation is of course chiefly dependent upon the precise nature of the etiological factor. In diphtheria the inflammation may take on the formation of a diphtheritic membrane. A somewhat similar process is seen in cases of thrush; streptococcus infection may also cause a membranous formation. In most of the acute infections the changes are of a catarrhal nature, as in scarlet fever, measles and the like, and the inflammatory changes may take on precisely similar lesions as those which occur in the skin or in the mucous membranes of the pharynx. In syphilis and variola the process may be manifested by the formation of pustules and the same may be found in phlegmonous esophagitis of any etiology. In simple catarrhal esophagitis the mucous membrane is hyperemic, covered over by a glairy mucous and the submucous connective tissue is edematous and swollen. More or less extensive erosion of the epithelium takes place, usually as a granular desquamative process like an exfoliation. Traumatic esophagitis is much more frequent in the lower animals than in man. It is a very frequent condition in herbivorous animals from the swallowing of bits of bale wire, hard and sharp twigs, and the like. It is a very frequent cause of death in domestic animals. The carnivora suffer similarly from sharp fragments of bone. In the human it occurs most frequently from bits of sharp metal, fragments of solder or tin, pins and the like, either swallowed by accident or taken with the food, however fish bones are among the most frequent offenders in this respect. The resulting lesion may be but an erosion of the mucous membrane with a subsequent inflammatory reaction, but abscess formation may occur in this manner as in a case cited by G. Goulet[4] in which a fragment of bone became lodged in the esophagus. The bone was removed and the resulting abscess drained, but nevertheless the patient died and at autopsy a purulent mediastinitis was found. This incident, as stated above, is much more frequent in the lower animals than in man. Modern methods of diagnosis and treatment by means of the operating esophagoscope render these occurrences now of less serious nature than formerly. In the esophagitis caused by heat or such irritants as highly seasoned foods, cantharides, or mustard water, a simple catarrhal inflammation ordinarily appears, but of course more serious grades may follow. In corrosive esophagitis such as caused by the strong escharotics as solutions of silver nitrate, iodin, the mineral acids and concentrated alkalies a direct destruction of the layers of the tube takes place. The depth of the lesion naturally depends largely upon the concentration and amount of the solution. In suicidal cases in which the amount taken has been large the erosion may include the entire walls. In two cases autopsied by the author, in one of which a concentrated hydrochloric acid was taken and in the other heavy sulphuric acid, the erosion has extended through the walls of the tube, coagulating the blood as it went and infiltrating down into the mediastinal tissues, so that even the aortic arch was blackened by the acids. Both cases were effectual suicides. Where the irritant or escharotic is in low concentration it may cause but a slight wrinkling or puckering of the mucosa, though in case of colored solutions as in Prussian-blue, tincture of iodin and the like, staining of the membrane will be diagnostically characteristic. Most of the powerful escharotics used for purposes of suicide give quite characteristic lesions readily recognized by the experienced pathologist; this is often assisted by a characteristic odor. Thus in carbolic acid poisoning the scar is white, leathery and dry and the odor is highly diagnostic. Glacial acetic acid produces intense edema, swelling and a jelly-like appearance with the typical odor of acetic acid. Hydrochloric acid gives off its odor and produces a blackened mucous membrane with very extensive surrounding coagulation of the blood into black clots. Sulphuric acid produces more extensive local erosions and its burns are also black, but the odor admits of little confusion with that of hydrochloric acid. Nitric acid gives its typical yellow, quite unmistakable coagulation. The strong alkalies, which on the contrary are rarely taken with suicidal intent, give a black greasy-feeling scar with liquefaction necrosis rather than the coagulation process seen in poisoning with the strong acids. If the patient survives these escharotic poisonings the diseased mucous and submucous tissues are cast off in large shreds and flakes; sometimes almost complete casts of the membrane may be extruded. At the points of eschar separation edematous granulation tissue appears and as this matures cicatricial contracture of greater or lesser degree is certain to follow. In cases not too extensive early and persistent treatment sometimes, with the assistance of subsequent operation may save life and even a considerable degree of integrity of the tube. Phlegmonous esophagitis, such as occurs in traumatic injuries from surrounding suppurative processes through rupture of an adjacent abscess into the esophagus, is a comparatively rare condition; fortunately so, for as a rule it is a very serious one, but treatment now possible through the esophagoscope has considerably reduced the mortality. J. Guisez[5] has recently reported four such cases, all of which were operated through this instrument. One patient died of septicemia after the operation, another required tracheotomy because of sudden laryngeal edema two months after apparent relief of the abscess, and the two remaining patients were cured.

ULCER

If we include under the head of ulcer of the esophagus all those instances in which erosion of the mucous membrane occurs as in the numerous forms of esophagitis, we must conclude that the condition is of very great frequency. If on the other hand we reserve this name for only such cases as preserve more or less definitely the course and appearance of peptic ulcers, the condition must be assumed to be comparatively rare. Tileston, however, records, according to Osler, 40 such instances and occasional cases are reported with an occurrence which seems to bear a direct relationship to the frequency with which esophagoscopy is practiced. Due to the rather low grade of sensitiveness of the esophagus it is well established that even quite extensive degrees of ulceration of the esophagus may exist without causing any symptoms which would be ordinarily noted by the patient; indeed practitioners who use the esophagoscope frequently usually comment as does Joseph Sheenan[11] on the great frequency of the condition and the infrequency of diagnosis except by the esophagoscope.

Etiology.—Peptic ulcer of the esophagus is apparently etiologically dependent in some degree upon obstructive lesions of the stomach as a result of which regurgitation of gastric contents into the esophagus may be assumed to occur with ease or frequency. In the minds of most clinicians an additional factor of injury of the mucosa is the swallowing of hard particles, and this condition is probably present in most if not all instances. Ulcers of this character as a rule present the classic funnel shape but may assume, particularly with infection of their borders, any size and form. The habitual use of extremely hot or very cold food materials may also be a factor of importance as in ordinary esophagitis. Undoubtedly most ulcers of the esophagus follow abrasions or injuries from the taking of hard or irritating food materials. As has already been stated those animals or persons who eat very rapidly and with incomplete mastication of their food suffer most seriously from this type of ulcer. The diffuse erosions of the esophageal mucosa such as follows the taking of acids, alkalies or other like substances either by accident or with suicidal intent should not be properly classed as an ulcerative process. The author had a case of a professional sword swallower whose esophagus showed numerous ulcerations, some acute, many old and entirely healed. They had apparently interfered but slightly with his comfort or profession. Tubercular ulceration of the esophagus is very rare except in generalized tuberculosis. Coplin states that when it occurs it is usually consequent upon perforation into the esophagus of outside tuberculosis processes, as, for instance, in tubercular lymphadenitis. Even in active generalized tuberculosis, tuberculous ulcers of the esophagus are very rare in the author's experience, which has been derived from an extensive postmortem service in a chronic tubercular hospital. In most of the cases seen the tubercular infection has apparently been secondary, engrafted upon primary traumata of the tube. Syphilitic ulceration of the esophagus is also a comparatively rare lesion and as in tuberculosis it appears to be in most observed instances usually an extension from extraneous luetic lesions. As also in tuberculosis, however, primary traumata of the tube may be followed in syphilitic subjects by the secondary development of syphilitic lesions. Even in gumma, as a rule, the process develops in outside structures, secondarily involving the esophagus which may, however, then present very extensive erosions. It is nevertheless first recognized when cicatrical contractures have begun.

Symptomatology.—Superficial, or slight, and ever some quite extensive ulcerations of the esophagus may produce few symptoms in many instances. Often the first symptom complained of by the patient may be those resulting from the cicatricial contracture of an ulcer in its healing process. It seems probable that symptoms are as a rule absent except where the process has penetrated as far as the muscle coats of the tube when spasm is very likely to occur. Pain on swallowing is one of the most frequent and characteristic symptoms and the patient is himself frequently able in these painful cases to precisely locate the point of ulceration. The bolus of food frequently makes a pause at this point. Tenderness over the sternum is sometimes present, but more frequently merely a sense of discomfort or of constriction is complained of by the patient. When the ulcer is located in the upper third of the tube, when perforation is imminent, tenderness and pain at a corresponding point along the course of the esophagus may be detected. Hematomesis may occur but it is rarely extensive unless associated with varicosities. Tickling, choking sensations and collections of mucus in the mouth are frequently evident and of course with all these symptoms is a distaste for food and a consequent emaciation which may even be of such degree as to strongly suggest carcinoma of the esophagus. Nausea and even vomiting is occasionally seen. In case of bleeding of any considerable extent blood will be found in the vomitus, usually not in great quantity and commonly of bright red color.

Diagnosis.—Diagnosis is most easily made and indeed in many instances possible only by the use of the esophagoscope. This instrument is practically indispensible since by it alone can we not only determine the existence and character of the ulcer but also differentiate it diagnostically from cancer. In many instances for this purpose it is advisable to remove a portion of tissue for biopsy. This method was particularly advocated by the late Dr. Harry Janeway. Sheehan speaks also very highly of the accuracy of the Einhorn string test, finding it positive in 65 per cent. of his cases. Except in extensive ulceration, notably in carcinosis, the x-ray diagnosis is usually neither very helpful nor accurate. Except by these methods absolute diagnosis is rarely possible though it may be made sufficiently accurate for therapeutic purposes from the symptomatology, from the observation of the act of deglutition in case the lesion is in the upper one-third of the tube, and from the history of the case which may in itself be very highly diagnostic, as in the case of the swallowing of a bit of oyster or clam shell, the swallowing of a bone which was immediately followed by pain and distress and subsequently by the other signs and symptoms of the condition.

Treatment.—Whenever possible treatment should be preceded by a thorough esophagoscopy. By this means direct application of such solutions as **argyrol, silver nitrate** or **iodin** may be made to the affected area. As a rule it will be found advisable to precede treatment by these agents with **cocain** or **novocain. Feeding** is accomplished preferably by the use of the **duodenal tube** and as a rule even when the stomach itself is in perfect health it will be found most satisfactory if the tube is first allowed to absolutely enter the duodenum and not to simply enter the stomach. **Rest**, preferably in bed, is advisable, though it is not absolutely necessary in all cases and some patients may successfully carry on considerable activities while undergoing the treatment. In some cases, as in extensive tubercular or syphilitic ulcers, gastrotomy and feeding through the fistula may be necessary. **Bismuth paste** combined with argyrol or with **magnesia** may be very helpfully used in many instances and when it is impractical or unnecessary to use gastric or duodenal feeding, in some instances successful results may be reached by the use of these materials alone, associated of course with soft non-irritating forms of food. Any indicated general treatment as in syphilis or tuberculosis should be fully carried out. Time is an important element in most cases.

Prognosis.—Very many patients get well spontaneously and of course prognosis is entirely dependent upon the nature, extent and location of the ulcer. Small ulcers as a rule need give little anxiety but it must always be borne in mind that cicatricial contractures after healing are likely to become of serious import and the early use of the esophageal bougie may be indicated.

STRICTURE

Obstruction in the lumen of the esophagus is among the most frequent esophageal conditions which the physician is called upon to treat. The subject admits of so many possibilities in diagnosis and so many secondary conditions may result from it that it is of paramount importance and interest, both diagnostically and therapeutically.

Etiology.—Stricture of the esophagus may result from many conditions or lesions within the esophageal wall itself or from extrinsic factors. Among the more frequent extrinsic factors concerned in the production of the condition are aneurysm, chiefly of the ascending and transverse aorta, tumors of the mediastinum; Hodgkin's disease, tubercular lymphadenitis, lymphoma and all other diseases of the lymph-nodes which may cause them to become enlarged; mediastinal or cervical abscess from any cause; and thyroid goiter or tumors and cysts of the thyroid. Extensive pericardial effusion may cause obstruction of considerable degree and in some instances of cardiac hypertrophy and dilatation the size of the heart may be so great as to cause sufficient compression to seriously inconvenience the patient. In some of these instances under the observations of the author, the patient has been obliged to lean forward in eating because of difficulty in swallowing when lying on the back. Because of the damage which may follow forceful dilatation these outside obstructions must always be considered, particularly in the dilatation of stricture of the esophagus. Where adequate x-ray and fluoroscopic facilities exist there is little danger now of serious error in the elimination of these extrinsic conditions which cause obstruction to the esophagus.

Cases of actual stricture of the esophagus may be considered either inflammatory or neoplastic in origin. Congenital stricture occurs in the infant occasionally but usually either cures itself in the subsequent growth of the child or the infant promptly dies either as a result of the stricture or frequently from some dependent or associated condition. Congenital stenoses are very unusual in the adult. One of the very most frequent causes of simple stricture of the esophagus is the previous swallowing, either accidently or through intent, of some erosive material which, having first caused inflammation or ulceration of the wall, in the subsequent healing of the inflammatory lesion is followed by stricture during the stage of cicatrization. It is frequently observed that months or even years may elapse between the infliction of the lesion and the appearance of symptoms indicating stricture, or the symptoms of the stricture may closely follow on the beginning of a healing process. Esophagitis of any type may be followed by such stricture, and cases are cited in which so apparently superficial a type of esophagitis as that in diphetheria or in thrush may be followed by serious stricture. In the contracture following even sharply localized ulcerations of the esophagus such as may occur from the swallowing of a foreign body, serious stricture may develop and it is well to bear in mind this fact in the preventive treatment of the condition, since the early use of the bougie may obviate very serious stricture, or local treatment through the esophagoscope may so promote early healing in destructive lesions of this kind as to obviate serious stricture thereafter. Stricture due to neoplasm will be considered under this immediate heading but it must be remembered that after forty years of age the most common cause of stricture is carcinoma and the burden of diagnostic proof after this period lies chiefly in the disproval of this condition. Syphilitic stricture is not frequent and in the author's experience when it occurs it is most commonly a result of extension of an outside luetic process, a gummatous one into the lumen rather than a true syphilitic ulceration of the esophageal wall primarily. Tuberculosis of sufficient extent and activity to cause serious stricture of the esophagus usually kills the patient before grave esophageal lesions appear.

Symptomatology.—The chief symptom in all forms of stricture of the esophagus is difficulty in swallowing. The degree to which this is manifest is to a considerable extent an indication of the degree of the stricture, though in nervous and hyperesthetic patients an accompanying esophageal spasm may seriously alter the valuation. It must not be expected that the degree of difficulty in swallowing will be constant at all times. In cases in which spasm probably plays little part there is still great variation from day to day in this respect and curiously some individuals will be able to swallow solids better than liquids or *vice versa.* Sooner or later more or less dilatation of the proximal portion of the tube takes place and as this increases greater and greater amounts of food may be stored in this space. As a result the patient complains of a sense of fullness, compression or actual pain at point corresponding to this dilatation; dyspnea may develop as a result and sooner or later this material will be regurgitated. While the stricture is still quite permeable this may take place at times, and at times not, depending very largely upon the character of the food taken, its degree of mastication and the rate at which it is swallowed. Acute lancinating pain is occasionally complained of in stricture but when present it commonly indicates that ulceration or acute inflammatory changes of some sort are also present.

Diagnosis.—There is little difficulty in the diagnosis of organic stricture of the esophagus. The symptoms are in themselves highly suggestive and absolute diagnosis is readily accomplished through fluoroscopy or by the x-ray plate. The absence of the characteristic splash or murmur of deglutition is helpful in some cases. Diagnosis by the use of the esophageal bougie is very conclusive in most cases, but the method admits of considerable danger especially when diverticula are present or when the walls of the tube are very friable as in carcinoma. As a general rule it may be said that it is the wiser procedure to fluoroscope or x-ray before the bougie is introduced and of course no force should be exerted in the passage of the bougie. Until the nature of the process has been fairly well determined it is far better to employ the blunt-pointed rather than the sharply conical bougie. In just so far as possible the nature of the stricture should always be determined before treatment is entered upon. This can be surmised in considerable part from the history of the case, as for example in the condition occurring after caustics of some kind have been taken. Usually also the x-ray plate and the fluoroscope will suggest definitely the nature of the stricture as mentioned above but in some instances it is further advisable to study the tube through the esophagoscope when in the case of neoplasm a quite characteristic picture will be presented which will also determine to a very large extent the character of treatment most advisable.

Treatment.—In some instances the lesion may be found to be due to the presence of a foreign body. This is usually determined through the **esophagoscope,** through which removal may be effected. Where the obstruction is due to a neoplasm, particularly if it be of innocent nature, removal is also occasionally possible through this instrument. The late Dr. Harry Janeway was particularly successful in operations of this kind which necessarily demand a high degree of technical skill and experience. When the obstruction is the result of external impinging bodies as enlarged nodes, abscesses and the like appropriate operations are demanded, depending naturally very largely upon the location of the tumor in particular. When stricture is caused by a greatly distended pericardial sac, medical or surgical treatment for this condition is indicated rather than attempts at the treatment of the stricture of the esophagus. Treatment must always be based on a thorough diagnosis not only of the location and degree of the stricture, but also in so far as possible on a study of its cause. Whenever a well-founded suspicion of a syphilitic etiology is found, the therapeutic test for this disease should be well tried out before efforts at dilatation should be attempted, whether a positive Wasserman reaction is obtained or not. If neoplastic masses compressing the esophagus are present, **deep x-ray** and **radium therapy** should be tried before dangerous operations are attempted, except of course where such delay would be a serious matter. Dilatation with the bougie should be performed only when as thorough an idea of the lesion and its nature has been determined as is possible. Where marked dilatation proximal to the stricture exists and particularly where diverticula are present, the greatest possible care must be taken in this method of treatment and unless the initial treatments are made under the fluoroscope the blunt-pointed instrument is preferable. The bougie must be well lubricated, usually with olive oil, and force must not be used until the operator is entirely sure of the ground when graduated sounds may be safely employed. Once a satisfactory passage has been accomplished it must be followed at not too infrequent intervals by systematic dilatation which would be done before symptoms indicating its immediate need are presented. When the stricture cannot be passed in carcinoma, the use of radium is now giving exceedingly good results, and just as soon as sufficient channel has been attained, the duodenal tube may be passed and left in place. Feeding by this route is preferable from every standpoint to that through a gastric fistula. Rectal feeding is of but definitely temporary value, and cannot be depended upon for tedious cases.

Pathological Anatomy.—Stricture of the esophagus from whatever cause usually is found either in the upper or in the lower third of the tube but it may of course occur anywhere along the course of the tube. This is due to the fact that all the conditions chiefly concerned in the production of stricture are most frequently operative in one of these two portions of the organ whether the cause be intrinsic or extrinsic. The character of the stricture will be entirely dependent upon the nature of the case. When due to ulcerations the contraction is usually limited in length, is narrow and irregular while the resulting superior dilatation is somewhat of the character of a diverticulum. When the elemental cause is some erosive condition, as from a diffuse esophagitis, the resulting stricture is likely to be long, narrow and tortuous, due to inequality of cicatrization. As a rule in stricture produced by extraneous lesion, as tumors, aneurysm and the like, the zone of compression is smooth and localized and esophagoscopy or fluoroscopic study will show the line of the mucous membrane to be smooth and regular.

TUMORS

Except for cancer, tumors of the esophagus are not frequent. Sarcoma is particularly rare and of a series of 175 cases of sarcoma of the alimentary tract collected by Corner and Fairbank but 14 were esophageal growths (quoted by Coplin). Other mesodermal growths are also very infrequent, though fibroma, myxoma, lipoma and myomata are occasionally reported. They are of negligible clinical importance, except that they may from time to time cause obstruction. Polypi of the esophagus are of this type. Papilloma and adenoma are also infrequent and are of little clinical importance. They are supposed to originate when present chiefly from aberrant tubular glands similar to those of the mucosa of the cardiac end of the stomach. Cancer of the esophagus is by all odds the most common form of primary esophageal growths and it is the only neoplasm of real clinical importance to the practitioner.

CANCER

Nearly all carcinomata of the esophagus are of the squamous-celled, epitheliomatous type. Of 54 esophageal cancers collected by Butlin, three were scirrhous, one medullary, one colloid and the remaining instances were all of the squamous-celled, epitheliomatous variety. They are therefore usually of slow growth and of a relatively low degree of malignancy. They may exist for a considerable period of time without producing striking symptoms or an observable degree of cachexia; in fact this last evidence of cancer may be entirely absent until extensive obstructive symptoms have appeared. As regards the point of usual appearance they occur as primary growths chiefly in the lower two-thirds of the organ. Kraus states that of 901 cases, 397 were in the lower third, 302 in the middle and 158 in the upper third of the tube, and 45 involved more than one part of the organ. They may be restricted to but a very small part of the organ or they may become widely diffuse. In the former instance unless by accident they are very likely to escape diagnosis until late. Many of them probably exist for years without detection as indicated by the history taken in conjunction with the postmortem findings, though Rolleston says that most patients die within one year after the appearance of symptoms. The time of detection naturally depends mostly upon the degree of obstruction which is manifested in the case. Ulceration is absent according to Coplin in 10 per cent. of the instances. In the author's clinical experience the disease is rarely diagnosed except in ulcerated instances; for unless ulcerated the symptom of pain is rarely prominent and large diffuse growths such as produce signs of obstruction are almost invariably ulcerated.

Etiology.—Only two factors seem to be definitely associated in the development of cancer of the esophagus,—age and sex. Fitz states that eight-tenths of the cases occur between the ages of forty and sixty, and one-third between those of forty and fifty. From 71 to 88 per cent. (Zenker and Von Ziemssen) are found in men. Present opinion seems to indicate that cancer rarely if ever develops from preëxisting innocent ulcerations, though some authors still state that there is an association of esophageal carcinoma with chronic irritations as from the use of greatly heated, very cold or highly spiced foods. Embryological defects probably bear no relation to the development of cancer of the esophagus.

Symptomatology.—As a rule the most frequent, most constant and earliest symptom is difficulty in swallowing. The patient will frequently be able to state with considerable accuracy the exact point at which the bolus of food or the drink meets with obstruction and fluoroscopic study of the act of deglutition will usually also definitely indicate this point. This symptom naturally varies very much in its degree and persistence; as a rule in cancer it becomes progressively more marked. Sooner or later pain is experienced in swallowing. This symptom seems to be more or less definitely associated with the existence and degree of ulceration, though it may be present in cases in which no ulceration is demonstrable. At first pain is present only as the bolus passes over the diseased area but as the case progresses it may be constantly present or it may be represented by a sensation of distention or discomfort which in cases unassociated with marked dilatation above the obstruction more or less certainly locates the point of the lesion. Regurgitation of food, sometimes mixed with blood or with fragments of neoplasm, may occur. Such fragments may be utilized for microscopic diagnosis as mentioned by many authors. Of recent note is the report of L. Timbale[12] of a case of sarcoma of the esophagus. In the experience of the author this is rarely practical, for the reason that such fragments are too necrotic for anything like satisfactory microscopic accuracy. Blood in greater or lesser amount may be regurgitated, or the initial hemorrhage may be very severe and even fatal. The usual absence of stomach contents in the material regurgitated is noteworthy. As the disease progresses distaste for food develops and sooner or later cachexia, greatly contributed to by insufficient alimentation, appears. Cough very similar to the cough of gastric disease appears in many cases and it is as a rule much aggravated by the taking of food or drink, even cough mixtures; it is relieved by vomiting or regurgitation. Eventually interference with respiration is noted and aphonia is a very common symptom as the recurrent laryngeal nerve becomes involved from extension of the neoplasm. Serious respiratory interference may appear either as a result of dilatation of the tube from compression of the pneumogastric nerve, or from erosion of the bronchi or trachea, which is very frequent in the last stages of the disease. As a general thing little tumor mass is likely to present itself in the neck, for the average case is really mostly intrathoracic, but where the growth involves the upper levels of the tube the tumor mass or the dilated tube itself may be present in the neck. Enlarged and invaded lymph-nodes, however, do appear in the neck even when the growth is of the lower one-third and, in practically all instances of advanced type, more or less distention of the neck veins is observable. Usually the suffering is greater when the growth is chiefly of the first third of the esophagus. It is obviously in such instances that the symptoms and signs of salivation become most annoying. It is usually accompanied by a foul odor and an escape of blood-stained exudate takes place. M. Loeper calls particular attention to neuralgic symptoms and cites cases in which a left cervical neuralgia radiating toward the jaw was present. He also calls attention to occasional spasmodic attacks of anguish or suffocation which may be confused with the distress of angina pectoris.

Diagnosis.—Diagnosis is not as a rule particularly difficult, though characteristic symptoms and signs may not appear in sufficient or in insistent enough a form to bring the patient to the physician until relatively late in the disease. Undoubtedly the most certain and the earliest definite evidence is furnished by the use of the esophagoscope, but the tyro must use care in its application; for in advanced cases serious damage may be inflicted. The same applies, though with less force, to the careless, unskilled or injudicious use of the bougie. In advanced cases the general appearance of the patient, the anemia, cachexia and emaciation and the probable discovery of enlarged lymph-nodes, either on inspection, palpation, or more readily by fluoroscopy or x-ray study, render diagnosis almost certain. In practically all instances in which the use of the esophagoscope is contraindicated positive fluoroscopic or x-ray plate diagnosis is possible. Where neither of these means is accessible the experienced clinician will not usually fail in a correct tentative diagnosis from the history, appearance, symptoms of dysphasia, obstruction and common-sense application of the "law of probabilities" because of the relative frequency of this condition as compared with others likely to be confused with it in persons of the usual age at which cancer occurs. Careful use of the bougie will usually assist in the demonstration of stricture and in the case of cancer, more or less blood-staining of the bougie will ordinarily be present which should not occur otherwise, except in ulcer. Sheehan lays great stress on the accuracy of the Einhorn string test. This may be readily enough performed by any practitioner, no matter how limited his facilities may be. The greatest difficulty in the diagnosis of cancer of the esophagus lies in the differentiation from extrinsic tumors which secondarily compress the esophageal lumen. Particular care must be taken in the differentiation from aneurysm. Tuberculosis of the bodies of the vertebræ, abscess, mediastinal tumors of any type, tubercular lymphadenitis, Hodgkin's disease and notably syphilis must all be considered. Where any doubt as to the latter possibility exists, even in Wasserman negative cases, the therapeutic test must be applied. Notwithstanding the great accuracy of diagnosis possible from the use of the esophagoscope, the clinician must not accept its findings as of absolutely conclusive nature, unless they are substantiated by the general clinical aspects. The author has seen cases in which a diagnosis of carcinoma has been made by men of undisputed skill and experience in the use of the esophagoscope which subsequently were shown to be instances of enlarged lymph-nodes with compression of the esophagus, and in one instance a syphilitic ulcer was diagnosed by this means as a cancer, a finding corroborated by the pathologist from the microscopic examination of a bit of tissue removed through the esophagoscope. Only those familiar with the difficulties of such diagnosis can appreciate the care that must be exercised in some instances. Such errors should emphasize the importance of associated clinical study and judgment. The author wishes to call particular attention to and to especially commend the attitude taken in regard to x-ray diagnosis by the writer on this subject in this volume (BAETJER, pp. 241-284).

Treatment.—Even where early diagnosis is made in cases of esophageal carcinoma medicine offers only palliation in the way of treatment. Surgery is little better; for its most brilliant results in these cases can be estimated as no more than delay of the inevitable. Happily on the other hand, **radium therapy** does give us a means of greatly alleviating these patients, of prolonging their lives and perhaps of actual cure, though the author confesses that he has personally seen no cases which demonstrate this as a scientifically proven fact. Sippy has recently devised a simple mechanism which will apparently greatly facilitate the accurate placement of the therapeutic radium and its use in association with the study of progress under the fluoroscope will greatly simplify and facilitate the method. C. W. Hanford[6] in a recent preliminary report cites 18 cases so treated and presents a system of dosage which at least gives a basis for future definite study, though Hanford still expresses himself as dissatisfied with its accuracy. He advocates an initial dose of 50 mg. and a time exposure of from eight to ten hours. The radium is then advanced up the diseased area about one inch and another exposure of eight hours made and thus on until the entire diseased area has been so treated. He recommends that a second treatment follow in from three to four weeks. He summarizes his conclusion as follows:

"Advance has been made, for in a previously always fatal disease, a percentage of patients are now saved. Dysphagia is relieved. A majority of patients are benefited and life is prolonged. Gastrotomy is avoided." Personal communications to the author of various radiotherapists who have been treating these cases lead him to feel that we now have in radium at least an agent of tremendous value in the treatment and alleviation of these cases. Time only can tell us if it is actually curative but certainly we are now justified in having hope in at least early cases.

X-ray treatment similarly benefits suitable cases very materially. It will often give great relief from the most distressing symptoms present and it certainly prolongs both comfort and life. Since it cannot be so directly applied as radium it is hardly likely that it will be as successful, except, perhaps, in an occasional case. Whether or not the radioactive substances are being used in the treatment of a case, great comfort and improvement in nourishment may be effected by the use of the **duodenal tube** for feeding purposes. It is a much safer, more comfortable and efficient means than the use of the stomach tube, particularly since it may be left in place for days at a time, and this without seriously annoying the patient.

Palliative surgery is employed now only in very exceptional cases, and, except where it is introduced to facilitate the use of the radio-active materials, it is of very doubtful kindness to the suffering patient. None-the less **gastrotomy** and feeding through the fistula is occasionally demanded, especially in those patients suffering from such serious malnutrition that radium treatment is not immediately possible. The humane aspects of any case should be considered before it is practiced merely to prolong a life of suffering.

In cases obviously fatal in termination **morphin** should be humanely employed; some patients are, however, sufficiently relieved by **codein**, by **luminal** and by the lesser hypnotics and sedatives. Rectal feeding may be temporarily utilized, but is much more efficiently replaced by duodenal feeding in most instances or when feeding is impossible by gastric fistula.

The author wishes to protest against the routine use of the **esophageal** bougie in those cancerous cases which present marked stricture. In his experience it increases suffering and rarely materially prolongs life, except that it be employed to facilitate radium application or the passage of the duodenal tube.

Prognosis.—Prognosis is wholly determined by the availability and success of surgical or radio-active treatment. Where these measures are unsuccessful the duration of the case rarely exceeds one year after the appearance of definite symptoms.

Pathological Anatomy.—Studies of the pathological anatomy of this condition indicate that the growth begins as an isolated, elevated and indurated area in the epithelial lining of the membrane. As it extends and grows its greatest diameter is usually upward and downward rather than about the tube, so that the initial stricture is generally monolateral rather than circinate. As ulceration and erosion take place—and as a rule the growth is inward rather than outward into the surrounding structures—, the mucous membrane presents a rough and no longer sharp line of demarcation, a fact which is of considerable diagnostic value in fluoroscopic study of the condition. When stricture results, as it ultimately does almost invariably the contracted lumen is rarely straight and regular but it is tortuous, irregular and rough. Extension into the surrounding structures ultimately takes place in all advanced cases. The adjacent lymph-nodes are naturally most frequently earliest involved and the recurrent laryngeal and pneumogastric nerve trunks may be invaded or compressed by the neoplasm with a consequent symptomatology. Erosion into the trachea or bronchi, into the arch of the aorta or into the venæ cavæ may take place, leading of course to a terminal hemorrhage. Similarly the bases of the lungs and even the heart may become involved. The mediastinal lymph-nodes as well as those about the larger bronchi are almost always more or less invaded in long-standing cases. Except when the original growth is of the upper levels the tumor mass presenting in the neck is not usually large. The picture presented in esophagoscopy in cancer of the esophagus is usually almost unmistakable to the trained observer.

DILATATION

Dilatation of the esophagus may be either diffuse, involving the entire course of the tube, or it may be localized in which instance a diverticulum may sooner or later develop.

Etiology.—Most cases are caused by organic stricture or appear as a result of spasm of the tube. There are possibly a few instances in which dilatation is the result of congenital defects. It has been observed, as stated by Fitz, in a seven-months' old child in which any other explanation seems improbable. Zenker and Von Ziemssen stress the possible rôle played in the condition by an enfeebled muscular contractility due perhaps to unrecognized general disease. Such cases are described by W. Oettunger and R. Gaballere[10] under the name of *mega-esophagus.* These authors report eight cases, four in considerable detail. They believe the condition to be not so rare, and explain the etiology of the condition as allied in pathogenesis to similar factors concerned in Hirshsprung's disease. Cade and Morenas[14] state that the condition may remain latent for a long time and note that it is discovered by accident largely or because of some complication which has arisen. When the condition develops as is certainly usually the case as a direct result of spasm of the esophagus or from the presence of obstructions to the lumen, that is, to stricture, there can be no doubt that its appearance is greatly accelerated by rapid eating and improper mastication of food.

Symptomatology.—The symptoms of dilatation of the esophagus are briefly those which result from the mass of the dilated tube when it is filled with food, and those of difficulty in swallowing. Regurgitation of often enormous quantities of material usually a short time after eating is frequent and the material so ejected has a very foul odor. The odor of the breath in many of these persons is very bad, and they suffer as a rule from a more or less constant sense of suffocation and poststernal distress. Aphonia may follow even in noncancerous cases. In some cases the contents of the dilated organ is not regurgitated *en masse*, as it were, but through occasional attacks of vomiting which are particularly excited by exercise or by emotional stress. Many of the symptoms present in stricture are naturally also present in dilatation.

Diagnosis.—Diagnosis is ordinarily easy. It is made most definitely on the detection of the overly filled tube, the regurgitation and vomiting, the sense of discomfort relieved by vomiting, the difficulty in swallowing and the constantly increasing discomfort and pain in the thorax and neck as food is taken. A longitudinal area of dulness, varying in size with the grade of the lesion and relieved by the vomiting or regurgitation, can ordinarily be made out, but the most conclusive evidence is furnished by fluoroscopy or by x-ray study. Not only does this make diagnosis of the condition positive, but it also gives at least a clue to the probable cause, locating at the same time the variations of the process. The method is quite indispensable to the study of the condition, for it indicates also to a very definite degree the need and type of treatment.

Treatment.—The initial thing to be done is to discover the cause of the dilatation. This will be found in most cases to be a stricture of some kind at least, or a condition due to a spasm either of the cardia or of some other portion of the tube. It is possible that obstructions lower down in the digestive tract, as in the pylorus, may induce secondary dilatation of the esophagus, but this is certainly an unusual finding. Steps should be taken as soon as possible to **relieve the causative factors**, but it will be found particularly difficult to study and dilate a stricture where marked dilatation of the esophagus has taken place. The tube should be washed out as thoroughly as possible. For this purpose water, normal saline or possibly mild solutions of permanganate should be used. When this has been accomplished attempts at dilatation may be made, but only with the direction of the fluoroscope or assisted by carefully taken x-ray plates. If, as will be the case in spasm, dilatation can be accomplished a **duodenal or Rehfuss tube** should be introduced and the subsequent feedings should be given by this means. Even where no difficulty is experienced in satisfactorily dilating the esophageal stricture as in spasm, nonetheless the feedings should be by tube so that the dilated muscle coats of the esophagus may be no further distended and an opportunity given for them to contract down to their normal status or as nearly so as possible. General treatment is very important in these cases even though in marked instances it is very largely unsuccessful. The general tonics, particularly **strychnin, iron and arsenic**, are to be given. **Quinin** seems to be beneficial in many cases. Drugs may be given hypodermically or through the stomach tube. Even for considerable periods after cause of the dilatation may have been removed the same precautions and treatment should be continued and when the patient is finally permitted to return to normal feeding he must be particularly cautioned as to rapid eating, and complete comminution of the food taken must be insisted upon.

Prognosis.—When the dilatation has been great, the prognosis as to recovery is wholly bad, though some of these individuals may with the assistance of good care and cautious living go on in tolerable comfort for a very long time. When in mild form and where the cause of the condition may be fully eliminated, even though the dilatation has never been extreme, if it has persisted for some time, complete relief is difficult of accomplishment and the patient must persistently observe great care in eating and particularly in mastication. He must in every instance make sure that one bolus of food has fully descended before another is taken and while large amounts of fluid at meals may somewhat increase the symptoms they seem to be advisable in considerable degree in the convalescent phases of the condition.

Pathological Anatomy.—The degree to which dilatation of the esophagus may proceed before it gives rise to imperative symptoms is truly tremendous so that the resulting cavity may contain as much as a liter (2 pints) or more of fluid or food. As a rule most areas of the tube are involved and a very irregular dilatation results, the caliber and shadow of which will be found frequently to vary greatly from time to time as it is studied under the fluoroscope. Occasionally, however, an aneurysmal-like dilatation causes the appearance, in case it be in the upper third, of a distinct tumor in the neck. The author is convinced that at least in a good many instances these saccular dilatations are not formed after the manner of true diverticula, but that they represent areas of decreased resistance in the esophageal wall, perhaps due to myomalacia or to a localized trauma of the wall or possibly to trophic lesions. When the entire tube is involved it pursues an irregular course, displacing itself as it distends, depending largely upon the position and pressure exerted by the more rigid structures which form its anatomical relations.

DIVERTICULUM

Although diverticula are in many instances developed under conditions similar to those in ordinary dilatation of the esophagus and although the pathogenesis is very similar in many respects the subject demands special consideration because of its great frequency and because of its clinical significance. But very little of any real importance, except in diagnosis and treatment, has been added in recent years to the conception of this condition as presented by Zenker and von Ziemssen in 1877. These authors divided diverticula into circumscribed protrusions of the esophageal wall caused by pulsion on the one hand, and by traction on the other. The lesion is found most commonly in middle life or in old age, and is rare in infancy or in childhood.

Etiology.—Pulsion diverticula are formed when for any reason organic or inorganic obstruction exists in the lumen of the tube, or when, due to some increased lack of resistance to pressure, a giving way of some localized portion of the wall takes place. A diverticulum, instead of a diffuse extension as in ordinary dilatation of the esophagus, is formed because some localized or circumscribed weakening of the walls of the tube exists at some point. This point of lowered resistance may be due to a congenital defect in the walls, usually in the muscle walls, though this is probably the least frequent form of pulsion diverticulum formation. It occurs at the point where, through some erosive lesion, as in ulcer from any cause, there has followed weakening of the wall through which a herniation of the inner membranes then takes place. The same result is produced when extraneous lesions have weakened an area in the walls of the tube, especially when this weakness exisits in the muscle coats of the organ. This determination of the point of weakness may be due to the same causes which produce the initial stricture or it may be due to independent factors acting at the same time. Pulsion diverticula are produced, however, not alone in instances of strictures. In some cases, beyond any doubt, where no stricture exists there is nevertheless a circumscribed weakening of the walls of the tube as from an ulceration, and the ordinary contraction of the tube, which Meltzer has so convincingly shown to be not an inert passage but an active physiologic viscus, causes a herniation which becomes more and more accentuated until a diverticulum is formed. Ulceration resulting from foreign-body trauma is particularly likely to act in this way. Pulsion diverticulum is most commonly found to take place from the posterior or lateral walls of the tube; it is least frequent of all from the anterior aspect of the wall. It is usually most frequent in origin at about the level of the cricoid cartilage, though the point of the subsequent sac may be found deep within the thoracic cavity. It is most commonly pear- or club-shaped and opens into the lumen of the tube sometimes through a narrow slit, and sometimes through a wide, open circular mouth, depending probably upon its mode of origin and upon the initial process concerned in its formation. When the long axis of the sac becomes of any considerable size it is usually downward in the direction of the esophagus. Occasionally the long axis or the sac when small may be directed up. In some cases the mouth of the sac is a linear slit which may be nearly or quite as long as the entire sac itself, giving a postmortem appearance suggesting at times a bilateral, double-barreled effect of congenital origin. Pulsion diverticula are usually single. As a rule the sac is lined more or less incompletely by a layer of stratified squamous epithelium similar to that normal to the esophagus; this is laid down on a submucosa of connective tissue mingled with more or less cicatrical tissue. The muscle investments of the esophagus are incompletely spread over the sac as a rule, even in small diverticula. In the observation of the author, ulceration of the wall is frequent though this is contrary to the findings of some writers. Although these diverticula may be of large size and may present in the upper levels of the esophagus directly beneath the skin, dermal perforation is rather infrequent unless the process has been caused by some originally external lesion of the esophageal wall.

Symptomatology.—Pulsion diverticula of small size may exist for a long time without causing in themselves any direct symptoms, though they may secondarily produce more or less disturbance. As a rule there is a rather close relation between the size of the diverticulum and its degree of symptom production. The first food swallowed may produce few symptoms except a sense of difficulty in swallowing and a sense of fullness in the neck. The filled sac may be visible or appear on palpation. In a good many cases after the sac has been filled, unless it be of large size, the subsequent food of that meal may be swallowed with comparative ease, though a sense of more or less suffocation is usually complained of. Cough sooner or later follows with the ultimate regurgitation of the material filling the sac, though rarely is all of it regurgitated at any one time and it is not as a rule ejected with the force frequently seen in diffuse dilatation of the esophagus. The ejected food may then be again swallowed, some of it perhaps passing into the stomach and some re-entering the sac. It is occasionally aspirated into the trachea. The entire contents of the sac is not usually thus ejected; some of it remains to putrefy and irritate. Food may be retained in these sacs, particularly in the large ones, for very long period of time. Sometimes the patient has found by experience that massage of the presenting mass in the neck will facilitate the emptying of the sac with relief of the symptoms. Sounds passed may enter the sac, or, if the opening into it is narrow, they may pass directly into the stomach. When a sound has entered the sac it is frequently possible by leaving it in place to pass another instrument along the normal channel or several sounds may first enter the sac very much as in the somewhat similar physical condition, divertula of the urethra. The symptoms become more and more insistent as the diverticulum increases in size until eventually it gives much distress. Emaciation appears, pain develops, fever from absorption from the infected and fermenting sac may appear and the patient becomes very uncomfortable or he may die from sepsis, from malnutrition, or from various terminal exhaustive states. Occasionally the regurgitation or vomiting may be almost paroxysmal in character. L. Bard[11] reports a case of an army officer who had been able to withstand the rigors of field service yet who had a period of vomiting at about four o'clock daily and whose chief complaints were of dyspnea, strangulation and aphonia. He was found on examination to have an esophageal diverticulum which pressed upon the trachea.

Diagnosis.—As a rule diagnosis is a very easy matter. As in so many other esophageal conditions the x-ray and flouroscope furnish the most accurate and satisfactory data, not only as to simple diagnosis, but also in information as to the size, nature and relations of the diverticulum. The method is invaluable. Although the use of bougies and sounds was formerly obligatory in diagnosis, with the x-ray and fluoroscope they are not usually necessary and their use is frequently attended with considerable danger, particularly in the hands of the inexperienced. Once diagnosis has been made it is better not to use these agencies unless for therapeutic purposes in the dilatation of a subjacent stricture.

Treatment.—Where the size, location and relations permit, the **sac should be operated upon.** A great many cases showing successful operation of this character are now on record and though the mortality and unsuccessful lists are still large, unsatisfactory results are commonly obtained in cases presenting great obstacles to operative technic, or in those which have been neglected, or in which the condition of the patient should suggest delay, if possible, until the patient's resistence may be built up, and sepsis minimized by a careful washing out of the sac through a catheter or the Rehfuss tube. Even when complete success is not achieved improvement can usually be expected and secondary operations may be performed advantageously. Where the sac is not too large considerable benefit may be achieved by feeding through the **Rehfuss tube** or by the use of the **duodenal tube.** Great benefit may follow **simple dilation of the cardia** in slightly advanced cases or in those in which operation is not possible. When the primary fault lies in a stricture of the esophagus or is elementally due to cardiospasm or esophageal spasm, treatment of these conditions will often give surprising benefit. Quite a few patients proceed with considerable comfort often throughout life, some without being aware of their condition. In almost all instances at least symptomatic benefit is reached by careful mastication, selection of small portions of food and by slow and deliberate deglutition. As a rule fluid and semifluid articles of diet are preferable in cases of diverticulum, but with these equally as with solid particles the amount of food taken at each mouthful must be small and great deliberation in eating must be practiced.

TRACTION DIVERTICULUM

Traction diverticulum is quite frequent. It rarely reaches a size anywhere approximating that seen in the form just considered. It is found most commonly projecting from the anterior or lateral aspects of the esophagus. It is frequently multiple and it originates from traction rather than from increased interior pressure. Its most common origin is through some inflammatory focus adjacent to the esophagus which has became attached to the wall of the organ, and, as contraction of the eschar tissue takes place, the area is pulled outward in a funnel-like projection forming a V-shaped pouch which extends out from the lumen of the esophagus. It occurs mostly in the lower levels of the esophagus near the bifurcation of the bronchi. Its usual origin is in or about necrosing or cicatrizing tuberculous lymph-nodes and it may naturally occur in any level of the esophagus. J. B. Hawes[7] has recently reported 2 cases of traction diverticula which perforated into the bronchi. The condition may develop after abscess formation anywhere in the neck and is apparently quite frequent after burrowing peritonsilar abscesses, and sometimes after spinal caries. These diverticula may form where some suppurative process has ruptured into the esophagus and from whatever mode of origin they are ultimately very prone to communicate through a perforation into surrounding spaces. They may thus rupture into the mediastinal space, into the trachea or large bronchi, or may even communicate through fistula perforating the skin. Their nature may be determined only by the detection of food substances which thus escape. Some authors believe that certain of these traction diverticula originate from fetal defects, connected in some way with the embryology of the bronchial clefts but this seems to be extremely doubtful and can pertain only to such as present themselves in the uppermost portions of the esophagus. Several diverticula of this type have been reported from war wounds, following rifle or machine gun wounds of the neck, or from fragments of high explosive shells. They may also originate from foreign body perforations of the esophageal wall with subsequent external scar formation or through tuberculous or syphilitic ulcerations. While rarely of the great size to which the other type of diverticula develop, they frequently cause quite as much annoyance to the patient and through their perforation may at any time excite the development of very serious secondary conditions. The symptoms are very similar to those of pulsion diverticulum: vomiting after eating, sense of weight back of the sternum, dyspnea, difficult deglutition and so on. Where no stricture of the esophagus exists the diagnosis by means of the x-ray or fluoroscope is much more difficult than in the other type of diverticulum for the reason that, due to the lack of increased intra-esophageal pressure and consequent failure of the tube to distend abnormally, the opaque meal may fail to enter the diverticulum and it thus escapes detention even though solid particles of food may give great annoyance. Some of these conditions heal spontaneously and many instances doubtless exist in which few symptoms appear. Where the diverticulum is located in accessible areas, **surgical treatment** is very successful and, even when surgical intervention is quite impossible because of the location of the lesion, healing may take place when feeding is accomplished for some time through the stomach or duodenal tube. In general the treatment is much as in ulcer and perforations of the esophagus. Early diagnosis is of particular value in the treatment of the condition.

RUPTURE

Rupture of the esophagus is a very rare and unusual accident even in such conditions as cancer, phlegmonous esophagitis or in ulceration. Although associated for many years with an active general hospital service in a great city, the author has never seen this accident in any traumatic condition. It may occur when extensive erosive disease of the esophagus has taken place and an attempt is made to swallow large masses of food, or, as stated by Osler, it may appear in violent vomiting. Even here it is a very unusual finding. Tears of the walls through the bases of ulcerations are not however particularly unusual especially when careless feeding or inexpert instrumentation is permitted in cases of stricture or ulceration. Perforation of the esophageal walls by the injudicious use of the bougie are sufficiently frequent to cause the wary physician to use this instrument with great care in those cases in which ulceration or necrosis of the wall is suspected.

Symptomatology.—When the rupture is a large one, sudden collapse takes place with the entrance of food into the mediastinum or pleural cavity. If death does not occur at once, septic processes are set up which usually quickly terminate life. Other symptoms are such as will develop from the specific area invaded.

Diagnosis.—In patients not known to have been suffering from a diseased condition in which weakening of the esophageal wall is a definite part, diagnosis is very difficult and in these cases in particular the patient often does not live long enough to permit of any extensive study. Fluoroscopy after an opaque meal makes diagnosis easy enough in most cases.

Treatment.—The only treatment is that of the symptoms and it is usually entirely without avail. Where the leakage is slow, as through the wall of an old ulcer, **surgery** may be useful, depending of course upon the location of the leak. The condition is practically identical in such instances with that in perforated diverticulum. **Catheterization** of the area with aspiration through the esophagoscope has been suggested, but the author is not aware of any cases in which this has been successfully employed.

HEMORRHAGE

Bleeding of slight extent from the esophagus is of very frequent occurrence. It occurs from slight abrasions of the mucosa and submucosa, due to the swallowing of abrading articles of food, particularly from pieces of clam or oyster shell and the like. It may follow an esophagitis from almost any cause, particularly from those which cause erosions of the muscous membrane. It is a common sympton of ulcer and it may be very profuse in some instances of this kind. Hemorrhage occurs in many forms of heart disease, especially in old mitral obstruction, in aneurysms or in other mediastinal or thoracic tumors which cause over-distention of the veins of the neck. It is probably most frequently seen in hepatic disease, particularly in cirrhosis of the liver in which varicosities develop in the veins of the esophagus as a result of the reversed venous circulation caused by portal obstruction. The size to which the veins of the esophagus may attain is so great as to cause more or less obstruction of the tube in cases of advanced liver cirrhosis, and also to but slightly lesser degree in some instances of mediastinal or thoracic tumors or even in advanced mitral disease. The loss of blood in instances of this kind may be so large as to be of immediate serious import, and to require transfusion. In nearly all cases blood may be found in the stools as well as in the vomitus since a certain amount of blood is likely to pass downward through the stomach. Very severe, and perhaps immediately fatal hemorrhage may appear in instances of perforated aorta or ruptured aneurysm. Slight hemorrhages may require no treatment except attention as to the point of origin of the hemorrhage. Cancer should always be considered. When hemorrhage is due to foreign bodies, these objects must be removed. Swallowing of very cold fluids may check considerable venous oozing; adrenalin may act similarly and solutions of iron, of tannic or gallic acid may also check oozing of considerable amount. Direct application of **styptic chemicals** through the esophagoscope may be indicated but in most cases except those due to ruptured veins, to aneurysm and perforation or to extensive necrosis in ulcer or cancer little more than **rest** and **rectal feeding** is usually necessary. In the severe hemorrhage which occurs in cirrhosis from the rupture or distended veins, absolute rest in bed, usually under **morphin,** rectal feeding, the use of the **Murphy drip** and the hypodermic administration of **thromboplastin** are usually all that is necessary. In some instances, however, **transfusion** is indicated. As a rule when hemorrhage occurs in these cases the general condition of the patient is such that the hemorrhage even if severe is of relatively secondary importance to the general condition. Considerable relief of temporary symptoms may follow the loss of blood. The same is particularly true of the rupture of veins in cardiac disease, for in these instances also the hemorrhage usually gives definite relief and is often beneficial rather than otherwise.

SPASM OF THE ESOPHAGUS

Spasm of the esophagus is a very frequent condition. If we include all degrees of it in our consideration, it is probably the most frequent esophageal disease with which the practitioner has to deal. In discussing the condition we must constantly bear in mind that the esophagus is not simply a passive tube through which the food is passed on its way from the pharynx to the stomach, but that it is an active hollow organ, subject to most of the disturbances which occur in the other hollow viscera. The condition occurs most frequently in young adults, but it is frequent in old age in those types of patients most prone to the disease and it has been recently reported by James Bruce[2] in an infant of three years. In the experience of the author it is very frequent in temperamental young children, and it forms a very probable basis of explanation for many instances of food revulsion which occur in adults as well as in children.

Etiology.—Undoubtedly the most frequent basis of esophageal spasm is some disturbance of the emotional balance. So commonly is this the true etiology that the clinician is in danger of assuming this to be the case in every instance and therefore other important and perhaps removable conditions may fail to receive prompt or proper recognition and consideration. It is very common, almost universal in some degree in hysteria, and in all probability it is closely associated with the phenomenon known as *globus hystericus*. It is frequent in neurotics of all types, in hypochrondriacs, in the insane and it may arise in the emotionally unbalanced at any time. It occurs in chorea, in vagotonia, in tetanus and is seen in its probably most exquisite form in rabies. It occurs as a result of auto-suggestion and in imitation neurosis. In a considerable number of cases it undoubtedly develops as a result of bad habits in eating, especially in those individuals who are in the habit of eating rapidly and of swallowing improperly masticated food. The author has long noted its frequency in his medical students and in business men who are obliged, or think themselves obliged, to eat their food hurriedly and during business hours. It is frequent under these same conditions among soldiers and other groups of men whose time and methods of eating are necessarily, or through fault of custom, hurried, casual and irregular. In persons of this character, unless the bad habit is forcefully broken, the condition becomes worse instead of better as time goes on and permanent dilatation of very serious degree may so develop. Unquestionably also it may appear as a result of reflex disturbances or stimuli. It is frequent in pregnancy and in some patients only during pregnancy. It is seen in various uterine and ovarian diseases, in chronic appendicitis, gall-stone, duodenal ulcer and in practically all forms of disease of the gastro-intestinal tract. It is associated with great frequency in gastric hyperacidity. Herman Meyer[9] attempts to explain its occurrence in association with gastric ulcer on the basis of the neurogenous theory of ulcer formation, assuming the relationship to be due to a hypersensitive conducting system. When its association with so certain a relationship as in stimuli is accepted, it seems rather unnecessary to demand a more definite association between gastric ulcer and esophageal spasm. It is very unfortunate that the frequent association with emotional instability of one sort or another is very likely to cause the clinician to frequently overlook etiologic factors of organic nature, the removal of which prevent in many instances the development of esophegeal dilatations of very grave degree. One must not forget in this respect the frequency with which spasm is due to actual lesions of the tube, ulcers, stricture or organic nature, even with early cancer. It is frequently the result of lodged foreign bodies, but perhaps even more so in neurotics with this obsession.

Symptomatology.—The severity of the symptoms produced in spasm of the esophagus is frequently more a measure of the primary causative factor than of the actual degree of severity of the spasm. To a very considerable extent hysteria and fright contribute very materially to an exaggeration of the symptoms. Difficulty in swallowing is usually at first chiefly complained of, but most striking, constant and it may be the sole symptom in some cases, is a sense of distress or distention in the region of the mediastinum which appears very soon after eating and which is relieved only when the bolus of food passes into the stomach or is regurgitated. In early or developing cases the attacks are as a rule intermittent, but they become more and more severe, and more and more persistent as the case proceeds if no relief of its etiology takes place. Serious inanition may follow. Very many patients, especially those in whom the condition arises as a manifestation of a neurosis, attribute the symptoms to cardiac disease and in some instances their story it so well borne out by statements as to dyspnea, cyanosis and the like that the clinician may be seriously persuaded toward that diagnosis also. Its close simulation to the esophageal symptoms which develop in pressure on the esophagus in aneurysm must not be overlooked. Usually there is no actual pain present, for Meltzer, Jackson and others have shown that the esophagus is far more sensible to tactile rather than pain sense, but the distress which appears thus immediately after eating may be very severe. A great effort and considerable time may be necessary before the patient is able to force the bolus through the hiatus into the stomach. The characteristic murmur normally heard when food enters the stomach may be entirely absent or altered to an unrecognizable degree. Immediate relief of the distress follows the entrance of the bolus into the stomach. It is noteworthy that the distress is practically the same in cases of spasm when the food taken is fluid as when it is made up of solids and almost invariably the spasm is much more marked when the articles of food is distasteful to the patient. Severe salivation may be present. In those cases in which vomiting or regurgitation takes place it is quite characteristic. It occurs suddenly, without an introductory nausea, during or soon after a meal and the entire mass filling the distended tube is likely to be eructated at one time. Immediate relief of all symptoms follows. The amount of food which may be retained in an esophagus which on fluoroscopy appears but within the high normal may be tremendous, so great may the acute dilatation become. As a rule, the longer the standing of the case the greater the amount which may thus collect.

Diagnosis.—Diagnosis is rarely difficult. It can usually be based with reasonable security on the history and symptomatology of the case: the nervous or emotional characteristics and the habits of the patient must likewise be taken into consideration. When vomiting takes place the material thus secured is very characteristic in that it contains no elements of gastric fluids. Fluoroscopy will usually definitely demonstrate the true nature of the condition and in the author's experience, it is far more satisfactory in these cases than x-ray plates, since the varying waves of contracture may thus be readily studied. The opening into the stricture is mooth and funnel-like, quite different from that in organic stricture, particularly in that due to carcinoma. Contra-indicating conditions having been eliminated by clinical and x-ray study of the case, the bougie may be passed and it will usually be found in uncomplicated spasm that normal or even large-sized bougies will finally pass, particularly if entrance is not obtained with violence. Before sounding one must always be particularly careful to fully eliminate outside sources of constriction such as may be caused by enlarged lymph-nodes, aneurysm and the like.

Treatment.—Treatment may be primarily divided into that designed for the immediate relief of the condition, and that directed both toward the removal of the basic cause and of the secondary conditions which have developed as a result of the two. Treatment must be introduced with a thorough study of the habits of eating and with an elimination of its faults. The food taken must be **masticated thoroughly** and only small amounts should be taken into the mouth at a time. A definite effort should be made to swallow slowly and the patient should preferably eat with others, thus feeling himself under an obligation to give way as little as possible to the nervous manifestations of the disease, and from a sense of decorum he refrains from gulping his food. This does not of course pertain to cases occuring in hysterical persons who often dominate their entourage through sympathy for their afflictions. If time lacks for a proper normal meal, the curtailment should be in the amounts of food taken, and not in the time alloted for mastication. Most patients do better if they dilute the food in the buccal cavity with fluids, preferably with water, but it must be remembered that even water if taken in large gulps or rapidly will often produce quite as insistent symptoms as solid particles. **Atropin** or **belladona** are highly recommended by some clinicians, taken preferably so that their maxium effects will be manifest at the time of the meal. The author has found **bromids, luminal,** and at times **chloral hydrate** administered from thirty to forty minutes before meals, more beneficial even in those cases which originate primarily from some definite lesion such as in ulcer. A preliminary dose of **milk of magnesia** or of **sodium bicarbonate** also acts very well sometimes. **Dilatation of the esophagus**, particularly of the hiatus or wherever the most frequent spasm occurs, gives great benefit and is probably as a whole the most efficient treatment for spasm, as much for its mental effect as for purely physical reasons. As a rule one gets better results with the use of **blunt-pointed** rather than conical pointed **bougies**, and the introduction of the instrument should always be slowly and deliberately accomplished or real injury may be done to the tube which will, as it were, add injury to insult in both physical and mental ways. Verbrycke[18] recently advises the use of the old-fashioned Plummer water dilator. The author has never tried it. In the use of the bougie the physician should first, if possible, assure himself that no real organic lesions exist. Where the condition is due to anxiety, imitation neurosis or to other emotional conditions, the treatment must be specially directed toward the removal of these, but one must constantly bear in mind the frequency with which the condition is dependent upon definite disease lesions as gastric hyperacidity, gastric atony, ulceration, or is associated with disease of the duodenum or gall-bladder. **Relief of an existing constipation** often gives complete cure. Throughout full appreciation on the part of the physician of the value of **suggestion** and **autosuggestion** in treatment must pertain, but the physician must be constantly on his guard lest he forget the frequency with which organic lesions also obtain in these cases. Treatment of the general fault or disease is very important in these cases, the correction of an anemia, treatment of neurasthenia, hysteria and the like. When resulting organic changes as diverticulum or dilatation of the esophagus has taken place, these conditions must be treated precisely as though not associated with spasm, and at the same time the treatment of the elemental condition must continue.

Prognosis.—Prognosis is usually good when no irremedial primary lesion is present and when the amount of resulting dilatation is not too great. A great deal depends upon the coöperation which the patient gives to the physician, as well as on the physician's skill, persistence and personality. Even long-standing cases with dilatation of considerable degree can be greatly benefited or clinically cured.

Pathology.—The spasms may occur in any portion of the tube, perhaps depending somewhat upon the location of the point of stimulus. It is seen most commonly in the upper and lower reaches however and when it occurs at the point of entrance into the stomach, which is one of the most frequent locations, it is spoken of as *cardiospasm*. It is by no means constantly present in the same area in the same patient, though as the secondary changes incident to the condition begin to develop, particularly dilatation and diverticulum, its point of location seems to become more constant. It is noteworthy that the points of most intense and most frequent occurrences are those in which contracture is normally most powerful and frequent. At least in its early phases it appears to be in a way but an exaggeration of a normal process. A certain degree of muscle hypertrophy develops in the segments affected and muscle degeneration may succeed this process. Dilatation of very severe and quite remarkable degree may follow persistent esophageal spasm. Diverticula may also develop. In most of the cases, even in those showing very extreme grades of dilatation, considerable variation in the size and shape of the dilated chamber will be found at different times in the same patient. As a rule the course of the distended esophagus becomes contorted or twisted. Perhaps this is also an exaggeration of the normal, since Jackson states that kinking and tortuosity of the esophagus is probably part of its physiological necessity in the prevention of regurgitation. In a discussion of the "Diaphragmatic Pinchcock in Cardiospasm" Chevalier Jackson[8] produces very striking arguments based on his extremely wide experience, indicating the probable action of this same mechanism in the production of cardiospasm. He states that out of hundreds of cases of cardiospasm which he has studied he has seen none in which the spasm at the hiatus was any greater than that which might be called a maximum normal. Following the long-standing spasm, all manner of secondary pathological alterations may appear in the esophagus, as inflammatory lesions, diverticula, ulcerations, atony, myomalacia and paralysis.

PARALYSIS

Paralytic conditions of the esophagus are very infrequently reported, but reasoning from analogy it is very probable that they are more common than generally appreciated, for the reason that they are most likely to occur with other and more impressive conditions which dominate the symptomatic picture. It is seen in diphtheria, usually in association with paralysis of the palate, in postinfluenzal conditions, and in alcoholic and lead poisoning. It occurs as a result of nerve-trunk pressure in tuberculous lymphadenitis, in Hodgkin's disease, in syphilis and the like. It is seen in central disease of the nervous system, as in paresis, cerebral syphilis, apoplexy and advanced arteriosclerosis. Transitory forms sometimes associated with preëxisting esophageal spasm have been reported in hysteria. It seems very probable that this condition may be etiologically associated in some instances of pulsion diverticulum. In the recognized cases at least the upper levels of the organ seem to be most frequently affected, but it is very probable that considerable degrees of paralysis of the lower third may often exist with but few symptomatic manifestations.

Symptomatology.—The most striking and suggestive symptom is difficulty in swallowing. It may develop suddenly or slowly, depending upon its etiology. The patient complains of being unable to force the bolus of feed into the channel, though, when it has engaged, it may slowly pass downward. The characteristic murmur audible in normal cases is of course lost and though a gurgling sound is present, the second esophageal sound is absent, and in at least some instances deglutition is finally effected, largely with the assistance of the neck muscles. A sense of pressure is complained of but pain is usually absent. Dyspnea may appear, especially if the patient attempts to swallow large masses in which case however the act of swallowing seems to be something more readily accomplished. Patients are likely to find that taking a fluid in considerable amount while eating may assist in the act of swallowing, though it may also increase a tendency to regurgitation.

Diagnosis.—Diagnosis is made from the symptomatic picture present, the latter usually being associated with some evidences of the etiologically concerned disease or factors, and frequently with paralytic conditions of other organs. The sound will pass readily, often too easily, and the clinician will find an undue lessening or resistance with the possibility also of considerable lateral movement of the point of the sound. Fitz stresses the value of rapid onset of sluggish swallowing as a highly diagnostic point.

Treatment.—Primary removal of the cause is to be attempted. In luetic cases specific medication obviously is suggested. In some cases of nerve pressure from adenitis or neoplasm **surgical relief** must be considered, but in many more instances of this type the early use of **radium** or **x-ray** treatment will give a better and safer result. The author has found that in at least some cases of diphtheritic paralysis, additional doses of **diphtheritic serum** will give relief even though the paralysis occur late in convalescence. The basic treatment of alcoholic and lead cases is obvious. When difficulty is experienced in feeding the patient in comfort, the **stomach tube** or the **duodenal tube** may be advantageously employed, and when a definite tendency toward dilatation is manifested these methods should be introduced before serious damage has occurred. **Strychnin, quinin** and other drugs of like nature may be given and **faradization** has been suggested.

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CHAPTER XI

ANAMNESIS, THE PHYSICAL EXAMINATION, EXAMINATION OF THE GASTRIC CONTENTS, EXAMINATION OF THE DUODENAL CONTENTS

By Sidney K. Simon, A.B., M.D., and Daniel N. Silverman, M.D.

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The Anamnesis

In the diagnostic investigation of the gastro-intestinal patient, the anamnesis is always to be looked upon as a highly important factor. While a complete physical examination, laboratory tests and radiological observations are each necessary elements, which are not to be neglected in the general scheme of examination, the ultimate interpretation of many digestive conditions will often be found impossible without an accurate and painstaking history. It is likewise essential that the method of obtaining the history be carried out in a systematic manner, and upon the basis of some definite prearranged formula. In this way only, can one be certain that essential data will not be overlooked. Most patients, and more especially dyspeptic victims, show a desire to relate their own history in their own way. It is perhaps best that they be allowed to do so, but only if it be understood that this constitutes merely the preliminary skirmish before the real inquiry into the essential facts of the history is begun.

In order to elicit the many-sided details of the history in the most comprehensive manner, one should be trained and grounded in the art of history taking. This is acquired only by patient and conscientious effort. The attitude to the patient during the taking of the history should be somewhat that of the lawyer who is engaged in quizzing an unguided or hesitating witness. The accuracy of the responses obtained will be found to depend to a great extent upon the keen insight and the intimate knowledge of the subject on the part of the questioner himself.

THE PERSONAL HISTORY.—Under this heading should be recorded the name, age, sex, occupation and social status of the individual.

Age and Sex.—Too much importance should not be ascribed to either age or sex as etiological factors in digestive diseases. Peptic ulcer occurs occasionally in the new born and has been recorded even in intrauterine life. The disease, while most common during the third and fourth decades, may be encountered at any age. Duodenal ulcer shows a decided predisposition for the male sex, while in gastric ulcer the proposition is about even. Carcinoma of the stomach is essentially a disease of later life, though it is well to remember that cases are occasionally recorded before the age of thirty. Men are only slightly more affected with gastric malignancy than women, according to the tabulated statistics gathered from many authentic sources. Chronic gastritis is probably more frequently met with among men than among women, though modern conditions of living have undoubtedly narrowed the disparity considerably. As a rule the reflex or functional type of dyspepsias occur more often in women. This is due in great part to the extra strain put upon the nervous system by the child-bearing period and the menopause. Visceroptosis with its manifold dyspeptic sequelae is likewise more frequent among women than among men in the proportion of about six to one. Nervous influences are by no means lacking in the male, however, as the records of the comparatively large toll of functional dyspeptics will show.

Occupation and Social Status.—Vocation displays a marked influence upon the origin and course of many digestive disorders. The comparatively large number of individuals whose work condemns them to indoor life, such as bookkeepers, stenographers, clerks, factory workers, etc., are more prone to the development of atonic states of the stomach and bowel than those who find employment which entails more active exercise in the open. The business man, likwise, whose daily routine is encompassed within the confines of a poorly ventilated office, frequently finds himself an early victim of dyspepsia. The same may be said of the society matron whose mornings, afternoons, and evenings are taken up almost exclusively with indoor amusements. The rich are prone to eat too much and exercise too little, and thereby suffer the consequences of a plethoric constitution. The poor on the other hand are largely restricted in the selection of their diet to coarse and indigestible foodstuffs which throw an extra burden upon the digestive apparatus.

Intensity of mental or physical effort can, if sustained over a long period, exert a marked influence upon the digestive tract. A close relationship has been shown to exist in this connection between persistent overexcitation or depression of the nerve centers and the clinical manifestations of peptic ulcer.

A considerable number of victims of peptic ulcer are recruited from the ranks of the overworked business or professional man, the responsible executive and the financier.

In the industrial field, the workers who are brought into intimate contact with the heavy metals, such as lead, copper and arsenic, or who are forced to inhale poisonous gases or fumes, suffer not infrequently from gastro-intestinal lesions of a varied nature.

THE PAST HISTORY.—A complete résumé of all previous illness or constitutional anomalies is an indispensable part of every gastrointestinal history. A record of any previous digestive upset is particularly to be sought for. Digestive disorders in the adult can sometimes be traced to a general lowering of the health standards initiated during the childhood period. The habit of irregular or rapid eating, of imperfect mastication or the employment of unbalanced diets, are tendencies which are frequently carried over into adult life to lay the foundation for future digestive trouble.

Moreover, attacks of socalled acute indigestion in the child may prove to be an early indication of a chronically diseased appendix or gall-bladder or other abdominal lesions. Likewise, a predisposition to constipation or diarrhea in the adult can be traced at times to an irregularity of bowel functioning which has had its development during the childhood period.

Among the infectious diseases typhoid fever is probably the most important in regard to its subsequent effect upon the digestive tract. In fully one-half of the cases of gall tract diseases, a history of a previous attack of typhoid fever can be obtained. Influenza also produces a large toll of digestive victims as an aftermath of the acute stage.

This was demonstrated quite conclusively in the experience derived from the recent great pan-endemic.

The character and ultimate outcome of previous surgical operations should be inquired into in minute detail. If needed, recourse should be made to the operative records. At least in some instances it will be found that laparotomy has been performed without sufficient justification or without affording the anticipated relief. Some highly neurotic individuals are made worse by operation, even though clear-cut indications may have existed at the time for surgical intervention. The nervous make-up of the individual should, in fact, be studied from every angle and the various tendencies he has exhibited in the past should be subjected to the closest scrutiny.

Habits.—An insight into the daily habits of the patient sometimes offers an important clue to the interpretation of a digestive lesion. A persistent tendency to overindulgence in food, alcoholic beverages, tobacco, coffee, or tea furnishes a basis for the development of certain organic as well as functional disturbances of the digestive tract. Overeating or the improper selection of diet constitutes probably the most common fault in this respect among dyspeptics. Irregularity in meal hours may be regarded as a close ally to the above. Unrestricted indulgence in alcohol leads eventually to a chronic inflammatory state of the stomach and intestines, and in some cases subsequently to cirrhosis of the liver. Tobacco exhibits apparently a definite selective action upon the functional mechanism of the digestive organs. A particularly refractory type of gastric hypersecretion and hyperacidity can often be traced to tobacco and particularly to cigarette smoking as a source. The over-use of coffee or tea likewise predisposes the individual to abnormalities of gastric function. The caffein content of these beverages, when taken in large amounts, acts as an irritant to the nervous system in general. Midnight indigestion and insomnia frequently result from the drinking of the after-dinner cup of coffee or tea.

THE PRESENT COMPLAINT.—The time of onset of the present complaint should be carefully elicited. Many patients date the onset of their trouble to a fresh outbreak of symptoms, when as a matter of fact upon careful inquiry the symptoms will often be found to trail back over a considerably longer period. The initial causative factor is always to be sought for. It must be remembered, however, that the patient is inclined at times to ascribe innocent causes for his digestive trouble which might prove to be merely coincidental.

An intermittency in the severity of the symptoms is a characteristic of many chronic digestive diseases. For no apparent reason the clinical manifestations disappear in whole or in part, with recurrence after a varying interval of time. This feature is especially prominent during the progress of a gastric or duodenal ulcer.

Dyspepsia is a term commonly used to denote any type of distressful sensation, which occurs in the stomach or upper intestinal tract during the progress of digestion. Included under the heading of dyspeptic symptoms are pain, heaviness, fullness, nausea, vomiting, eructation, regurgitation and abnormalities of appetite and thirst.

Pain.—From a diagnostic standpoint, abdominal pain constitutes by far the most important of the dyspeptic symptoms. The first step in the investigation of the source of an abdominal pain should be directed to an explanation on the part of the patient of its exact character. Is the pain which he experiences cramp-like, aching, boring, gnawing, lancinating or merely a painful empty sensation in the epigastrium?

Next in order is the location of the pain, and in this connection the patient should be asked to point out the exact area covered by the pain, using his index finger as indicator rather than the entire hand as is so frequently done. The line of direction which the pain follows becomes of considerable value also in helping to determine its source. More than one type of pain is sometimes experienced by the patient, and if so, an attempt should be made to determine the character and location of each. The relation that abdominal pain bears to food or fluid intake is always of the greatest importance for the purpose of the history. Does the taking of food or fluid into the stomach increase the distress and, if so, how soon after eating does the pain appear? If, on the other hand, the patient is found to be relieved by eating, attention should be directed to the type of food or drink which produces this effect. In the same manner the fact that an alkali relieves the distress often furnishes a valuable insight into the nature of the underlying trouble. If relief is brought about by vomiting, the evidence may be considered conclusive that the presence of food in the stomach is a direct causative factor. In many cases the onset of the pain cannot be ascribed by the patient to the ingestion of either food or drink. This point is in itself of considerable value.

Furthermore, the relationship of abdominal pain to other factors besides the ingestion of food should be sought for. Among these factors might be mentioned the effect produced by physical exertion, posture, hot and cold applications, external pressure upon the abdominal wall, emotional excitement or depressive mental states. The evacuation of the bowels or bladder, the menstrual function, respiration or other normal physiological agencies, also exert a remote influence on abdominal pain at times.

When the pain is paroxysmal in character, one should think of the passage of stones or inspissated masses along the gall tract, genito-urinary tract, or pancreatic duct. Intestinal colic, Dietl's crisis, epigastric hernia, as likewise vertebral and spinal cord lesions sometimes give rise to intense paroxysmal pains in the upper abdomen.

Heaviness and Fullness.—These symptoms are usually closely associated and constitute a very common form of complaint on the part of the dyspeptic. Heaviness signifies the sensory reaction of a pathologically hypersensitive gastric mucosa to the weight or pressure of ingested foodstuff, while fullness expresses a similar response in terms of quantity or volume. The character of the food and the manner of its preparation constitute important factors in the production of these symptoms. Heaviness and fullness, appearing within a short while after eating, is present in most cases of gastritis as well as in benign and malignant ulcerations of the gastric mucosa. When these symptoms occur several hours after eating, or in the fasting stomach, a functional origin is to be suspected.

Nausea and Vomiting.—When nausea and vomiting are complained of, the patient should be interrogated upon several points. Does the sensation of nausea appear immediately after eating or only after food has remained in the stomach several hours? Does vomiting follow the nausea, and is relief obtained by the bringing up of the retained food? When vomiting relieves abdominal pain, it is almost certain that the cause resides in the stomach itself. Does nausea and vomiting exist independently of eating? An affirmative answer points strongly to a reflex source.

The character of the vomitus should always be looked upon carefully. Do the food particles which one finds ejected come solely from the previous meal, or does the vomitus consist of substances eaten many hours or perhaps days before? Is blood present, and if so is it bright red or dark in color, resembling perhaps coffee grounds?

The amount of the ejected material is another point that calls for investigation. A large quantity of vomitus suggests gastric stagnation and especially so, if food particles are discovered from preceding meals. A voluminous vomiting of clear gastric juice indicates, on the other hand, a reflex or a tabetic origin. A distinction must also be made between vomiting and regurgitation of food. The latter is always in small amounts, and is unaccompanied by retching.

The type of vomiting sometimes furnishes evidence of the underlying causative factor. The morning vomiting of the alcoholic addict, for example, is characteristic. So, likewise, is the early morning vomiting of pregnancy. In the former, the vomited material is composed usually of thick stringy mucus and there is no attending nausea, as a rule. Typical projectile vomiting is indicative of a cerebral tumor. When dizziness precedes nausea or vomiting, a cerebral lesion also is suggested or perhaps the cause will be found in some ocular disturbance (refractive error) or in the ear (Menière's Disease) or again in the sinuses. Some neurotically inclined individuals induce vomiting artificially in order to relieve their gastric distress. This may degenerate into a pernicious habit, which is frequently found difficult to cure.

Eructation and Regurgitation.—These rather common clinical phenomena are brought about by a disturbance of the mechanism of the cardiac sphincter of the stomach. Under normal conditions, the cardia remains closed during the entire period of gastric digestion. When, through extrinsic sources, the normal tonus of the sphincter becomes inhibited, a patulency of the sphincter develops, which is accompanied by the ejection of the chyme into the esophagus. Eructation or simple belching represents a reflux of the stomach contents into the lower end of the esophagus, the gas alone rising to the throat. Regurgitation, on the other hand, denotes the propelling of fluid stomach contents directly into the throat and mouth. Both symptoms have a common origin and may be present in all irritative lesions of the gastric mucosa, or they may occur as a result of a reflex from other abdominal organs. A purely neurotic type of belching is also commonly met with. Aerophagia, or the constant swallowing and eructation of air, is an aggravated form of the latter. Pain in the upper abdomen or in the precordium which is relieved promptly by belching can be ascribed usually to cardiospasm.

The Appetite.—The appetite is readily impaired when the digestive functions become upset from any cause. The lower animals wisely refuse to eat when sick. This represents Nature's method of staying the further progress of disease. This is only partly true, however, in man since the appetite or desire for food is in many instances artificially pampered. More or less complete loss of appetite is present in malignancy of the stomach, in chronic atrophic gastritis, in general tuberculosis, in mental depressive states, and in certain forms of gastric neurosis (hysterical anorexia). An increase of appetite (polyphagia) is frequently noted in diabetes mellitus, hyperthyroidism, and in gastric hyperacidity. A distinction should be made between a lack of desire for food (anorexia) and a fear on the part of some patients of the consequences which attends eating (sitophobia). This fear of eating is prominent in many cases of peptic ulceration. Bulimia is a term used to denote a grossly exaggerated or insatiable appetite. Individuals afflicted with this condition consume enormous quantities of food and drink. The etiology is not always clear. The desire for abnormal types of substances (pica) is found in neurotic individuals and usually indicates a low-grade degree of mentality.

Thirst.—Thirst plays a relatively small rôle in digestive diseases. In xerostoma large quantities of fluid are consumed in an effort to relieve the abnormal dryness of the buccal mucosa. In depletive diseases, such as chronic diarrhea or dysentery, the thirst is also greatly increased, as a rule. Dilatation of the stomach with marked stricture at the pyloric sphincter is usually accompanied by thirst, caused by a lack of water absorption. In the absence of the above conditions, the consumption of large amounts of liquid points to the presence of diabetes mellitus or insipidus as the most probable cause.

The Physical Examination

In view of the close and intimate relation that exists between diseases of the abdominal organs and other organs of the body, a complete physical examination of the patient is always essential, beginning at the head, so to speak, and terminating at the lower extremities. No detail is to be omitted, and for that reason it is best that the observations as a whole be undertaken in accordance with some definitely formulated plan of procedure. Inaccuracies arising in connection with the diagnosis of abdominal diseases can often be traced to a careless or a slovenly conducted physical examination.

The preliminary details should be arranged with the view of comfort for the examiner and the patient. The preferable position is the dorsal decubitus. It is an advantage, however, during the course of the examination, to place the patient in various positions, the right and left lateral, erect, and even in some instances in the knee chest posture. For this reason, the examination table should neither be too high nor too low, and should be so placed as to permit of ready accessibility from any side. A good light is essential for proper inspection, and the direct rays should be made to fall preferably over the left shoulder of the examiner. The various regions of the body to be examined should be freed of all clothing or other encumbrances, such as trusses, adhesive straps, plaster or other bandages.

The confidence of the patient can be gained at the outset by a friendly reassuring manner on the part of the examiner. During the process of palpating the abdomen, relaxation of the muscles can be accomplished in great part by entering into some light topic of general conversation. Also the hands of the examiner should be warmed before approaching the patient.

A complete physical examination of the alimentary tract includes every detail that may be obtained through inspection, palpation, percussion and auscultation of the various digestive organs.

MOUTH AND THROAT.—As the opening portal to the digestive tract, the mouth and throat bear a close relationship to all the other organs of digestion. It is here where foci of infection in great part originate, which in many instances lead to subsequent inflammatory states of the stomach, intestine, appendix, liver, gall-bladder and pancreas.

A strong reflected artificial light with head mirror should always be used in inspecting the mouth and pharynx. The size and consistency of the tongue is first to be noted. A flabby moist tongue with indentures of the teeth visible along the edges is present in many low-grade adynamic states, and particularly in chronic malaria. On the other hand, a small sharply pointed dry tongue with red margins is frequently found in typhoid fever and other prolonged infectious diseases. An inflamed denuded raw beef type of tongue is commonly associated with chronic dysenteries and diarrhea, and is also seen in pernicious anemia. The red angry tongue of sprue is characteristic, as likewise the flabby, heavily coated, raw edge type seen in pellagral cases.

The coating or fur on the dorsal surface of the tongue is made up of denuded epithelium and other debris. A thick coating is seen in starvation or when the patient is on liquid diet, also in acute and chronic gastritis, in chronic alcoholics and in intestinal stasis.

The examination of the teeth and gums follows next in order. Infectious conditions in the gums and around the teeth are recognized as fruitful sources for systemic infections in general. When suspicion of such infections exists after a cursory survey the patient should be referred to a competent dentist for further expert advice and treatment. Suspicion alone may rest upon the presence of carious or filled teeth and especially crowns and bridges and upon the existence of inflamed and purulent marginal gum edges. Absence of necessary teeth or poorly fitting artificial sets give rise to imperfect mastication with its attending ill-effects upon the digestive tract.

Inflammation and ulceration of the buccal mucosa is produced by local infections in conjunction with disturbed conditions of the digestive tract, undernourishment and systemic poisonings. The tissues of the mouth are ordinarily resistant to infection. Various discolorations of the mucosa and gingival surfaces are observed in plumbism, copper poisoning, Addison's disease and pernicious anemia. The buccal mucosa and tongue are characteristically pale in all forms of anemia.

The throat should be examined carefully for any evidence of inflammation or ulceration. A history of pain or other irritative state in this region should lead to further inspection of the naso-pharynx, larynx and sinuses. A dry and injected pharyngeal surface suggests digestive disturbance as a probable cause. The tonsils should be subjected to special observation in every case. It is a good plan to draw the tonsil forward and attempt to express the secretion from the crypts. If any cheesy or purulent material appears, the patient should be referred, preferably to a specialist, for more definite diagnosis of pathological changes and for treatment, if necessary.

ABDOMEN.—*Inspection.*—With the abdominal surface fully exposed, and with the patient placed in a relaxed prone position, observation should first be made of the size, shape and contour of the abdomen. The shape and contour is largely determined by the congenital make-up or habitus of the patient. The long narrow thoracic abdominal body type (habitus asthenicus) is distinctive of congenital visceroptosis. Here one finds a markedly obtuse epigastric angle, a narrowed space between costal margins and ileal crest, frequently a detached tenth rib and a general loss of tone of the abdominal musculature. A broad or widely spaced abdomen, on the other hand, with well-developed musculature signifies usually an inherited robust constitution. Lack of muscular support in the abdominal wall is made apparent by a certain flabbiness upon palpation, and by a prompt bulging of the lower aspect when the patient stands erect.

Under normal conditions with the patient in dorsal decubitus, the upper half of the abdomen, above the umbilicus, presents a slightly retracted or concave appearance, while the lower half is distinctly bulging or convex. Any variation from this type is significant of some abnormality produced either by an increase in the amount of gas or fluid, or by the enlargement of one or more of the underlying organs.

Considerable information may be derived from inspection of the cutaneous surface of the abdomen. The presence of linea albicantes furnishes evidence of a marked reduction in weight or else the existence of a previous pregnancy. A diffuse infiltration involving the abdominal wall as a whole is caused by a surplus accumulation of fat or by edema. Dilated or tortuous veins indicate some form of portal obstruction. In addition, search should always be made for hernias of the abdominal wall. Epigastric hernia may be productive of considerable distress and is not infrequently overlooked on inspection.

Visible pulsation of the abdominal aorta is present in many neurotic individuals with thin abdominal walls. This condition must not be confused, of course, with true aneurysm of the abdominal aorta or its branches, which is exceedingly uncommon.

In emaciated individuals the normal peristaltic waves of the stomach and intestine may be seen at times, especially when distended with gas. When some form of obstruction occurs, the peristaltic waves become more evident and appear as distinct prominences on the abdominal surface. These waves travel usually in the direction of the obstructed area, although antiperistaltic waves are likewise observed, especially in the obstructed stomach. In pyloric stenosis accompanied by marked dilatation of the stomach, the gastric contractions are sometimes observed below the pelvic rim. Difficulty may be encountered in distinguishing between the peristaltic waves originating in the stomach and those produced by an obstructed bowel. In the latter, however, the waves are more diffuse and are usually more distinctively vermicular in type. A gurgling sound signifying the escape of gas through a narrowed lumen may sometimes be heard at the height of the contraction. Any circumscribed bulging or shadows on the abdominal surface should be carefully observed from various angles. A small tumor can sometimes be detected in this manner to better advantage by inspection than by palpation. The size, shape and general appearance of an apparent tumor mass should be noted in detail, and an account should also be taken of its dependence upon respiration or change of position, as well as the influence exerted by the presence of food or fluid in the abdomen.

Palpation.—Palpation follows inspection in the natural order, and frequently serves to verify and amplify the facts previously brought out by inspection. Complete muscular relaxation in the dorsal position is essential for satisfactory results in palpating the abdomen. The right hand of the palpator should be placed flat against the abdominal surface and not at right angle, simulating the position taken during piano playing, as is so frequently done. For deep palpation the left hand can be made to override the right. While firm steady pressure is sometimes necessary, the patient should under no circumstances be made to suffer discomfort from the examination, since this calls forth resistance on the part of the musculature. It is best to begin with gently stroking or superficial palpation, covering each area of the abdominal cavity in turn. Much valuable information can be gathered from a superficial survey conducted in this manner. Indeed, deep palpation sometimes tends to push an abdominal mass away from the palpating fingers.

When tumors are present, heed should be taken of their size, form, position, consistency, surface characteristics, as well as their sensitiveness to pressure. The mobility exhibited by a tumor during respiration is also of assistance in determining the organ to which it is attached. A small tumor connected with the stomach or the intestine can be made to disappear in some instances by inflating with gas these organs from without.

Rigidity of the abdominal muscles (defensé musculaire) furnishes a valuable sign, pointing to some form of irritation beneath the surface. Complete rigidity of the wall, when not under voluntary control, is an almost certain indication of general peritonitis. Circumscribed rigidity furnishes equally positive information depending upon a localized peritoneal irritation or inflammation. The diagnosis in both instances is rendered more certain if sensitiveness to pressure is present in connection with the rigidity.

Localized areas of sensitiveness occur whenever inflammation or ulceration is present in the underlying structures. Caution must be used, however, in estimating the significance of painful spots located in any section of the abdomen. Neurotically inclined individuals are very prone to complain of sensitiveness to pressure even upon superficial palpation. When the mind is distracted from the immediate object of the examination the painful spot will often be found to disappear promptly. A true sensitiveness based upon definitely existing pathologic conditions is usually fixed at a specific area and is constantly present under all conditions.

Regional Palpation.—The order to be followed in palpating the various regions of the abdomen is optional with the examiner. The author has found it preferable to begin in the right hypochondrium and proceed successively to the epigastric region, the left hypochondrium, the left inguina, the right inguina, and finally to the umbilical and suprapubic areas. The lumbar region can be palpated to the best advantage by placing the patient in a partial lateral position, turning first to the right and then to the left. The bimanual palpation of the lumbar regions is greatly facilitated by taking a position behind the patient.

The Right Hypochondrium.—In this region one is enabled to palpate the enlarged liver, the gall-bladder, the pyloric aspect of the stomach, the right kidney, the hepatic flexure, the transverse colon and tumors connected with the omentum and retroperitoneal space.

When palpating the liver, note should be made of the consistency and surface aspects of the organ, the character of the edge presenting, whether smooth or rounded, as well as the presence of nodules or tumor masses.

The normal gall-bladder cannot be palpated, but the pathological gall-bladder frequently presents itself as a rounded mass overlapped to a certain extent by the liver edge. It is important to remember that large gall-bladders usually drop backward when the patient lies in a prone position and may therefore be palpated at some distance from the normal site at the level of the ninth cartilage. The over-distended gall-bladder is usually distinctly globular in shape and occasionally its fluid contents can be made out by fluctuation. The author has never been able to palpate stones in the gall-bladder, as has been claimed by others.

The normal pylorus may be palpated at times during contraction when the abdominal wall is thin and in a relaxed state. Pyloric tumors are circumscribed as a rule, are frequently hard and nodulated and as is the case with the liver and gall-bladder move readily with respiration.

The right kidney, under ordinary conditions, is more accessible for palpation than the left. The simple detached right kidney is a very common physical finding. Diffuse enlargements and tumors of the kidney are comparatively rarely encountered in the course of abdominal palpation. The movability of the kidney is not influenced by the act of respiration. The same is true of masses connected with the other retroperitoneal structures, as well as tumors attached to the transverse colon or the omentum.

The Epigastrium.—Here are encountered tumors or other masses connected with the pylorus, the lesser curvature and other portions of the stomach, the right and left lobes of the liver, the transverse colon, the small intestine and the omentum. A collection of fluid in the lesser omentum, subphrenic abscess, and a thickened dilated aorta or abdominal aneurysm are occasionally met with also in palpating the epigastric region.

The Left Hypochondrium.—In certain cases, where the stomach has become retracted, tumors of that organ may be encountered entirely to the left of the median line. Among other structures present in the left hypochondrium may be mentioned enlargements of the pancreas, the spleen, the splenic flexure, the descending portion of the colon, the left lobe of the liver, the left kidney and masses connected with the small intestine and retroperitoneum.

The spleen and left kidney can be palpated to better advantage with the patient in the left lateral position. An enlargement or tumor of the spleen may be recognized by the characteristic edge and the general form and shape of the organ, as well as by the mobility displayed during respiration. Pancreatic growths are immobile and present themselves for palpation usually to the left of the median line.

The Left Inguina.—From the standpoint of the gastro-enterologist, the descending colon, the sigmoid and the rectum are the principal structures to be considered in connection with the left lower abdomen. Growths originating in this section of the bowel must be differentiated principally from those connected with the pelvic organs and the genito-urinary tract. Hyperplastic enlargements or tumors of the uterus, left ovary and tube may be recognized mainly by their distinctive characteristics as determined by bimanual palpation through the vagina or rectum, while genito-urinary lesions are eliminated by careful study of the urine, cystoscopy and radiography.

The Right Inguina.—Similar deductions may be drawn in regard to the pelvic and genito-urinary organs in considering the right inguinal region as has been described for the left. In addition one encounters, in this important section of the abdominal cavity, the appendix, the cecum, the ascending colon, each with their special pathologic anomalies. A mass in the right lower abdomen gives rise at times to considerable difficulty in diagnosis. One must think not only of the pathologic appendix when confronted with such a condition, but also of the possibility of a simple inflammatory state or of a tuberculous, malignant or syphilitic lesion affecting the ileo-cecum or ascending colon.

Fecal masses can usually be ruled out by their doughy feel, freedom from sensitiveness as a rule, and their prompt disappearance after the patient has been given a strong purgative.

The Umbilical and Suprapubic Regions.—The small bowel rarely comes under observation during palpation, owing to the fact that inflammatory swellings and tumors of this section of the bowel are comparatively infrequent. Any abnormal mass in this region is usually derived from enlargements of the pelvic organs or from an over-distended or diseased urinary bladder.

Percussion.—Percussion of the abdomen in most instances provides little information that cannot be better obtained through inspection and palpation. The lower border of the stomach can sometimes be mapped out by percussion when the patient is placed in an erect position, after swallowing several glasses of water. It is seldom that one is enabled to distinguish the size or the position of the stomach, however, by percussion alone. There is little difference to be noted in the tympanitic tone elicited over the distended stomach as distinguished from that produced over the bowel.

Percussion, however, proves of considerable service in determining the presence of free fluid in the abdomen, as well as in locating and outlining abnormal masses and tumors. When fluid is present in the abdominal cavity, the percussion note varies with the position taken by the patient. Under such circumstances the line of dullness will be found to follow closely the level of the fluid, being most marked, as a rule, in the flanks and in the inguinal regions. An exception may be noted when peritoneal adhesions are present, which interfere with the free transference of the fluid. The presence of free fluid is also determined by gentle bimanual tapping with the fingers of each hand placed upon opposite points on the abdominal wall. In this manner a fluctuation wave is produced that is quite characteristic. This must be differentiated from the false fluctuation sometimes produced by an over-collection of fat in a relaxed abdomen.

Auscultation.—Auscultation occupies a relatively unimportant place in abdominal examination. It is sometimes used in conjuction with percussion in locating the size and position of the hollow viscera. With the bell of the stethoscope placed in the region of the stomach, gentle tapping or a scraping movement of the finger will produce a clear resonant note which makes it possible in some instances to locate the borders of the stomach. This method is not, however, to be relied upon. Occasionally friction sounds synchronous with respiration can be heard over the liver or spleen in connection with a perihepatitis or a perisplenitis. When present, these signs possess a definite diagnostic significance.

Auscultation of the deglutition sounds produced in the esophagus, first described by Meltzer, may prove of value for detecting the presence of an obstruction at the cardia. The bell of the stethoscope is placed in the costal angle beneath the ensiform cartilage and the patient is made to swallow a large mouthful of water. The first sound, which is of a squirting character, is heard almost simultaneously with the act of deglutition. Five to 10 seconds later a second sound exhibiting a bubbling or metallic character becomes audible as the water passes through the cardia into the stomach. The second sound is heard more constantly than the first. When stricture of the esophagus is present, there is usually a delay in the time appearance of the second sound, or else either one or both sounds may be entirely absent.

Examination of the Gastric Contents

THE NORMAL GASTRIC JUICE.—The gastric juice is the product of the mucosal glands of the stomach. This secretion is a watery, colorless fluid, holding in solution hydrochloric acid in the proportion of .2 per cent., the enzymes, pepsin, rennin and lipase, certain inconsequential mineral salts and a variable quantity of mucus. An admixture of duodenal contents with bile is occasionally present. The quantity of pure gastric juice secreted in twenty-four hours varies between 1,000 and 1,500 c.c. The total quantity of the secretion as well as the relative amount of acid or of enzymes present depends upon several factors. During the resting or inter-digestive phase, the secretion of gastric juice is markedly diminished or ceases entirely. According to the observations of Carlson, there is more or less continuous secretion of gastric juice even in the entire absence of food. The amount, however, is small, varying from a few cubic centimeters to not over 60 c.c. per hour.

The secretion of gastric juice is to a marked extent under the influence of psychic stimuli. This socalled appetite juice is called forth by seeing, smelling or even thinking of savory food on the part of the individual. The original observations of Pavlow on dogs and those more recently of Carlson on man would seem to emphasize the importance of this element upon the entire field of digestion.

In the fasting stomach a residium of approximately 50 c.c. is present, consisting chiefly of gastric mucus, swallowed saliva, and mucus from the respiratory tract along with small amounts of gastric juice.

Clinical Methods of Studying the Gastric Juice by Means of Test Meals.—THE TEST BREAKFAST.—In order to obtain accurate results in the conduct of the test breakfast, certain fundamental standards and rules must be rigidly observed. The meal should be given only on the fasting stomach. For this purpose, it is necessary that food and drink be prohibited for at least twelve hours preceding the test. A standardized meal only is to be given, consisting preferably of two slices of ordinary white bread, each slice one-half inch thick, and two glasses of water. This constitutes the socalled Ewald test breakfast, which should not be modified, as is wrongly done in some quarters by the substitution of other food substances. The contents should be withdrawn in exactly one hour, and the examination of the specimen carried out according to a definite formula in the manner to be described later. In some instances, where rapid gastric motility is indicated the contents of the test meal may be withdrawn from one-half to three-quarters of an hour after ingestion. The Ewald test breakfast performed in this manner yields sufficient information for ordinary clinical purposes, and is to be preferred as the method of choice in the daily clinical routine. When further insight into the behavior of the gastric secretion is required, recourse may be had to the method of fractional analysis introduced by Rehfuss in 1912.

THE REHFUSS OR FRACTIONAL METHOD.—A special stomach tube was devised by Rehfuss for this purpose. The rubber tubing is of small caliber, not over one quarter inch in diameter, to which is attached an olive-shaped metal tip, with large fenestrated openings. The tip is swallowed by the patient in a fasting state, and after reaching the stomach, the contents of the organ are aspirated and set aside for further analysis. The patient is then fed the ordinary Ewald test breakfast, and at the end of each fifteen minutes, 10 c.c. of gastric contents is withdrawn and placed in properly labeled glasses. The specimens thus obtained are later examined separately in the usual manner of the ordinary test breakfast. By this means, one is enabled to form an estimate of the entire phase of gastric secretion, including a survey of all possible variations. The fractional method has been found to be especially useful in the study of the gastric achylias.

A delay in the appearance of hydrochloric acid or of the enzymes is often noted during the earlier phases of digestion, extending over a period perhaps of one or two hours. This socalled false achylia might easily escape detection, unless the fractional method of gastric analysis is employed.

THE EXAMINATION OF THE ASPIRATED TEST BREAKFAST RESIDUE.—After aspiration the test breakfast should be examined in turn, macroscopically, chemically and microscopically.

The Macroscopic Examination.—The macroscopic inspection reveals many important facts. The quantity, color, odor and gross appearance of the contents are first to be noted. The volume of the Ewald test breakfast (450 c.c.) is reduced to a maximum of not over 120 c.c. at the end of the one-hour period. The average residuum obtained by means of the stomach tube under normal conditions is 75 c.c. An amount in excess of 120 c.c. indicates either motor delay or gastric hypersecretion. Similarly, when less than 30 c.c. is withdrawn it points to either hypermotility or a decrease in secretion.

Abnormal color may be due to the presence of certain foodstuffs, blood, bile, pus or may result from contamination with vegetable moulds or chromogenic bacteria, yielding distinctive discolorations, usually greenish. This green color is sometimes confused with bile stain.

The odor of the bread and water mixture of the test breakfast is quite distinctive, that is, mildly acidulous and bready. Excessive fermentation or admixture of foreign substances produces a change from the normal odor.

The amount of mucus present in the stomach contents is of great importance for the diagnosis of gastritis. A distinction must be made between mucus swallowed from the upper passages, and gastric mucus. The latter is clear and transparent, only slightly viscid, and never appears in clumps, as is the case with swallowed mucus. Again stomach mucus is intimately mixed with the bread particles, while the respiratory mucus impregnated with air bubbles floats to the surface.

Under normal conditions the gastric chyme is a homogenous mass, the various food products'having undergone complete maceration and mixing as a result of the churning movements of the stomach musculature. Poor chymification, therefore, is an indication of abnormal function, and is caused mainly by a deficiency of hydrochloric acid secretion. In achylia gastrica, the test breakfast is usually returned with the bread particles in almost the same state as when swallowed.

The Chemical Examination.—Qualitative and quantitative tests for the determination of free and combined hydrochloric acid, the total acidity, and the various enzymes of the gastric juice, furnish valuable information in regard to the functional activity of the stomach.

Qualitative Tests for Free Hydrochloric Acid.—A variety of color reagents or indicators are employed for the detection of free hydrochloric acid. Blue litmus is converted into red in the presence of all acids. More specific indicators for the mineral acids are solutions of congo blue and dimethylamido-azobenzol (Toepfer's reagent). Congo blue is changed to red in the presence of free hydrochloric acid and the yellow Toepfer's reagent is likewise turned red by the acid.

The *Gunzburg test solution* is not affected by organic acids and is quite accurate for even small amounts of hydrochloric acid. This test is carried out as follows: Two or three drops each of filtered gastric contents and Gunzburg's reagent (composed of phloroglucin gram 1, vanillin grain 1, alcohol 30 c.c.) are placed in a porcelain dish and evaporated to dryness. In the presence of hydrochloric acid a bright red crystalline deposit forms on the outer border of the dried-out precipitate. Care must be taken to apply the heat slowly, as otherwise the red color will be converted quickly into a burnt brown. A mixture of *resorcin and cane sugar* in alcohol constitutes another highly specific test solution for the mineral acids, first recommended by Boas. This reagent is prepared by adding 5 grains (0.324 gram) pure resorcin and 3 grains (0.195 gram) cane sugar to 100 c.c. of 94 per cent. alcohol. The technic of this test is as follows: 5 to 10 drops of gastric filtrate are mixed with from 2 to 5 drops of the resorcin solution in an evaporating dish. Heat is applied gently as in the phloroglucin-vanillin test. If free hydrochloric acid is present a bright vermilion red discoloration appears which is dissipated quickly upon cooling.

Quantitative Tests for Free Hydrochloric Acid.—Any of the above indicators may be employed in the titration of the gastric filtrate for the estimation of free hydrochloric acid. Toepfer's reagent is usually selected for the purpose. To 10 c.c. of the filtered gastric contents, 2 or 3 drops of Toepfer's reagent is added. A red color denotes the presence of free hydrochloric acid. The solution is then neutralized by the addition, drop by drop, of a solution of one-tenth normal sodium hydroxide, contained in a buret. The point of neutralization of the acid is marked by the conversion of the red Toepfer's reagent to a canary yellow. A reading of the number of cubic centimeters of decinormal sodium hydroxide solution employed indicates the amount of acid present. This amount is usually multiplied by ten in order to bring the reading to a basis of 100 c.c. of filtrate. The normal reading of free hydrochloric acid varies between 30 and 40. A reading above 40 would signify hyperacidity, and under 30 a subacidity.

The H-Ion Concentration Method of Determining Free Hydrochloric Acid.—The H-Ion concentration of any solution is the amount of ionized hydrogen contained in each unit volume. For the determination of the amount of ionized hydrogen in a solution two methods are available, either the electrometric or the colorimetric. For ordinary clinical purposes, the colorimetric method, though somewhat less accurate, is to be preferred because of its simpler technic. A considerable number of dye substances have been suggested for use as indicators in carrying out the colorimetric test. For the determination of the H-Ion concentration of the gastric contents, Clarke and Lubs selected thymolsulphonpthalein (thymol blue) as the most suitable indicator. It was found by these authors that the varying degrees of acidity of the free hydrochloric acid content of the gastric juice could be expressed more uniformly and with greater accuracy by the employment of this particular dye. Accepting this suggestion as a basis for further work in this field, Scholl and King were the first to devise a simple colorimeter, which could be employed clinically in determining the H-Ion content of the gastric juice.

Method of Scholl and King.—A series of standardized color solutions are prepared in sealed glass containers which represent varying degrees of H-Ion concentration. It has been found that these colors fade in the course of time, so that a renewal of the solutions may be required. Comparison with standardized colors should be made at least every twelve months.

Add two drops (.04 c.c.) of a .1 per cent. alcoholic solution of thymol blue from a graduated pipette to 2 drops (.04 c.c.) of filtered gastric contents. It is essential that the gastric filtrate be filtered several times, if necessary, or centrifugalized in order to clear it of any turbidness. A change to a reddish color denotes the presence of free hydrochloric acid. The shade of color reaction is now compared with the standardized color solutions, and a reading is taken, which expresses the degree of H-Ion concentration corresponding to the amount of free hydrochloric acid present. A reading of 1.8 to 1.4 represents normal acidity. 2.4 to 1.8 denotes hypoacidity and a reading of 3.0 is equivalent to anacidity. A figure less than 1.4 expresses hyperacidity. Should the specimen of gastric contents possess a greater H-Ion concentration than 1.6, 2 c.c. of distilled water should be added for dilution along with 2 additional drops of the indicator. The reading is then multiplied by 2 to include the degree of dilution.

As a result of a series of comparative tests and in conformity with the opinion of others, the authors believe that the H-Ion concentration method is capable of a greater degree of accuracy than is the case with the ordinary titration methods.

Estimation of the Combined Hydrochloric Acid.—Combined hydrochloric acid is estimated by means of a solution of Alizirin (alizirin sodium monosulphate in a 1 per cent. aqueous solution). This indicator is sensitive to the presence of free hydrochloric acid, acid mineral salts, and organic acids and is converted into a violet color in an alkaline medium. Ten cubic centimeters of gastric filtrate containing 2 to 3 drops of alizirin solution is titrated with N/10 sodium hydroxide until the violet color appears. This reading subtracted from the total acidity gives the amount of combined acid present.

Estimation of the Total Acidity.—The total acidity of the gastric contents is made up of free hydrochloric acid, combined hydrochloric acid, the acid mineral salts present in the food, and organic acids. To determine the total acidity, 2 or 3 drops of a 1 per cent. alcoholic solution of phenolphthalein is added to 10 c.c. of gastric filtrate. The same filtrate employed for the determination of the free acid may be used. Complete neutralization of all the acid elements is indicated by the appearance of a reddish color. The curve of total acidity runs a fairly constant and parallel course with that of the free acid. The normal reading of total acidity varies between 50 and 60. In hyperacid states records above 100 have been noted.

The Gastric Ferments.—The ferments, pepsin and rennin, are only active when free hydrochloric acid is present in the gastric juice in a sufficient amount to give a hydrogen-ion concentration above 3.0. The absence of hydrochloric acid renders both enzymes entirely inactive.

THE DETERMINATION OF PEPSIN.—*The Mett Method.*—This test yields fairly accurate results and is the one commonly employed by a majority of clinicians and laboratory workers. The Mett pepsin tubes are prepared by drawing fluid egg albumin by means of suction into finely spun capillary glass tubes. The albumin is then coagulated by boiling for five minutes and after cooling the tubes are hermetically sealed with paraffin. Care should be taken to exclude air bubbles and to have the albumin content fill the tube completely.

Before testing for pepsin, the tubes are first cut into smaller segments ranging from 2 to 5 cm. in length. These are placed in the filtered gastric contents which is kept at body temperature in an incubator for 10 hours. The hydrochloric acid content of the solution should approximate .18 per cent. At the end of the 10-hour period, if pepsin is present, the coagulated egg white is found to be digested in whole or in part. The digestive process begins at the ends of the test tube and extends progressively inward. Measuring the length of the digestive column in squared millimeters furnishes a fairly accurate method for estimating the degree of peptic activity. Should the digestive column measure 2 mm., for example, the unit of peptic digestion would be calculated as 4. One pepsin unit constitutes the amount of pepsin capable of digesting 1 mm. of egg protein contained in a Mett tube within a period of 10 hours. This is predicated upon an acidity of .18 per cent. free hydrochloric acid. When the acidity is above this figure dilution of the gastric filtrate is required, ranging from one-fourth to one-twentieth. The degree of dilution must be taken into account in the final calculation of the peptic activity of the fluid. This is done by multiplying the squared millimeter length by the diluting figure.

Ricin Test (*Jacoby-Some Method*).—One gram of ricin is dissolved in 100 c.c. of a 5 per cent. solution of sodium chlorid. This is filtered and 2 c.c. of the filtrate is mixed in a small test tube with ½ c.c. of tenth normal hydrochloric acid solution and 1 c.c. of diluted stomach contents. The ricin is deposited but as a result of the action of the pepsin clears up when the solution is kept at body temperature for 3 hours. One pepsin unit represents the amount of gastric juice which is sufficient to clear up 2 c.c. of the above 2 per cent. ricin solution in 3 hours at body temperature. Normal stomach contents contain about 100 pepsin units per cubic centimeter. The details of this test are rather intricate for quantitative estimations and the results are not as reliable as those obtained by the Mett method.

The Gelatin Test.—Five c.c. of a 3 per cent. solution of gelatin is poured into a glass test tube 15 mm. in diameter, and sterilized at 100° C. (237.3° F.). A vertical position of the tube is maintained until the gelatin becomes solidified by cooling. Five c.c. of the filtered gastric juice is now layered over the gelatin, and .02 gram of thymol is added. Digestion is allowed to proceed at room temperature for a number of hours.

A millimeter scale made of paper is pasted on the outside of the test tube, which serves as a measure of the degree of digestion taking place in the gelatin within a definite period. The total amount of gelatin digested may be read from the paper millimeter scale. Normal gastric juice digests, on an average, 2½ mm. in 24 hours. To insure accurate estimations, a number of tubes should be used in each test, and an average of the readings taken.

THE DETERMINATION OF RENNIN.—Neutralize 10 c.c. of filtered gastric contents with decinormal sodium hydroxide solution. The same quantity of previously neutralized boiled milk is added and the mixture is placed in an incubator. The curdling action of rennin should begin within 15 minutes. A mass coagulum usually forms when the mixture is allowed to stand for over 30 minutes, when sufficient rennin is present.

SIGNIFICANCE OF OCCULT BLOOD IN GASTRIC CONTENTS.—A gross amount of blood in the stomach contents is always indicative of some definite pathological lesion. Streaks or small traces of blood on the other hand possess no significance, particularly in cases where the stomach tube is used, owing to the frequent occurrence of slight abrasions of the mucosa during its passage. For this reason a positive occult blood test in stomach contents should always be interpreted with care, and no definite clinical inferences should be drawn based upon this finding alone. A negative reaction, however, is of considerable value in ruling out the presence of hemorrhagic lesions.

Tests for occult blood in gastric contents are conducted in the same manner as in the stool. The *benzidine test* is the most sensitive and is to be recommended for routine use. The technic is as follows: As much benzidine as will go on the tip of a pocket knife is dissolved in 2 c.c. of glacial acetic acid. The filtered stomach contents in about equal quantity is added to this solution along with a few drops of hydrogen peroxide. A promptly appearing, distinctly blue reaction discloses the presence of blood.

Compressed tablets of biborated benzidine, first introduced by Dudley Roberts, are very convenient for use and may be used instead of the benzidine powder. A small segment of the tablet is placed on a clean glass slide and dissolved in a few drops of glacial acetic acid. To this, 3 or 4 drops of gastric contents are added and mixed in well. In the presence of blood a blue discoloration appears almost immediately.

When benzidine is not available, the *Guaiac test* may be employed. This is carried out as follows: 10 c.c. of gastric contents is mixed in a test tube with 2 c.c. of glacial acetic acid and 15 c.c. of ether to dissolve out the hemoglobin. This mixture is shaken well and the ether is allowed to separate. The ether is then decanted into another test tube, and treated with a second mixture composed of 10 drops of freshly prepared tincture of guaiac and 30 drops of pure hydrogen peroxide. The presence of hemoglobin is indicated by a blue discoloration.

The microscopic examination of the test breakfast residue is sometimes undertaken in special cases, but because of the amount of undigested bread present is not usually satisfactory. The various features of the microscopic findings of gastric contents will be discussed in detail under the head of the examination of the motor meal residues.

MOTOR TEST MEALS.—The most satisfactory method of determining the motor function of the stomach is by means of special test meals, the principle of which is based upon the fact that certain foods of definite character and in measured amounts leave the stomach within a known specified time. A large number of such test meals with a varied combination of foodstuffs have been suggested in the past. The authors have been led to restrict their selection, however, to the test meals described below. These are regarded as sufficiently comprehensive for ordinary clinical purposes.

The Test Breakfast.—The Ewald test breakfast is given primarily with the object in view of testing the gastric secretions. Nevertheless, as previously mentioned, the recovery of an excessive quantity, above 120 c.c., is to be regarded as suggestive of a retardation of the emptying time of the stomach. In the face of this finding, a more definite motor meal test should always be instituted.

The Raisin Test.—The patient is requested to eat one or two tablespoonsful of seedless raisins or currants at some definite period during the day, preferably late at night or during the early morning hours. The raisins may be taken in conjunction with other food substances as, for example, bread, cake or puddings. The stomach contents are withdrawn six hours later. The finding of hulls or other remnants of the raisins in the stomach contents at the end of this period points to a definite delay in motility.

The Riegel Test Meal.—This consists of 400 c.c. of beef broth, about 150 grams of cooked meat, 50 grams of mashed potatoes and a good slice of wheat bread. The stomach is aspirated in from four to seven hours and the amount and character of the recovered meal, if any, is noted in detail.

The Authors' Test Dinner.—This constitutes a type of dinner meal, both in character and amount, which is partaken of daily by the average individual and serves therefore as a satisfactory index of the motor capacity of the stomach under normal physiologic conditions. The meal is given at 10 P.M., following a previous fast at least six hours, and consists of vegetable soup, partially cooked rice, a green vegetable, two slices of raw tomato, a small portion of meat (this may be omitted), bread and butter, and a baked apple with raisins. At 9 A.M. the stomach tube is introduced and the entire amount aspirated is set aside for further examination. The stomach is then lavaged until the water returns clear. The various washings are examined carefully for the presence of any solid particles. The findings of any element of the meal, either upon gross or microscopic examination, indicates a marked degree of gastric stagnation, pointing usually to pyloric stenosis as the cause.

Examination of the Gastric Residue After a Motor Test Meal.—MACROSCOPIC.—*The Gross appearance.*—In the more advanced types of gastric stasis the aspirated stomach contents separate upon standing into three layers. The top layer is composed of a frothy scum, caused by the gradual rising of the fermentation gas bubbles to the surface. The second layer is essentially fluid in character, but is usually cloudy and viscid, due to an excessive quantity of mucus. The bottom layer of sediment is made up of the more solid substances, consisting in the main of undigested food particles and other detritus.

Quantity.—The quantity of the gastric residue removed after a motor test meal will depend upon the character and amount of food eaten, as well as upon the time of withdrawal. In the more extreme types of stasis, resulting as a rule from pyloric obstruction, as much as one to two liters may be withdrawn after a twelve-hour fast. In minor degrees of delayed motility, on the other hand, due to simple muscular atony, the stomach will often be found to have emptied completely within seven hours after an average meal. Between these extremes various grades of motor insufficiency are encountered, depending upon the type and the severity of the etiological factor.

Odor.—The odor of the stagnation gastric contents is usually distinctly fermentative in character. When yeast cells and sarcinae are the predominant organisms, the odor is that of a sour must, very much like fermented bread dough. The decomposition of the protein element in the food material results in the production of hydrogen sulphide and other gases, yielding characteristic fetid odors. Likewise, a malignant growth of the stomach, which is undergoing disintegration and sloughing, may give rise at times to a distinctly foul odor in the gastric contents.

Color.—The color of the gastric residue partakes largely of the character of the food eaten. A copious hemorrhage into the stomach produces a dark brown or tarry black discoloration. The recovery of a dark coffee ground gastric sediment is commonly associated with the diagnosis of carcinoma, though a similar sediment may occur in benign hemorrhagic lesions as well. A green discoloration is produced by admixture with bile and also in some cases by the chance presence of certain chromogenic bacteria.

The Chemical Examination.—The filtered gastric residue obtained after a motor test meal may be titrated for the presence of both free and total acid in the same manner as described for the test breakfast. The hydrochloric acid and the enzyme secretion continue as long as any food material remains in the stomach. The total acidity is usually found to be high in the stagnant stomach contents, owing to an increase in the amount of organic acids generated. Etherized extracts of the gastric filtrate will yield distinctive odors in the presence of some of the fermentative organic acids such as acetic, butyric and valerianic acids.

Lactic Acid.—The generation of lactic acid in stomach contents is the result of the action of certain bacteria upon carbohydrates. This group of bacteria is only active when stagnation of food is present, and when the gastric secretion is deficient or lacking in hydrochloric acid. The occurrence of lactic acid in stomach contents, when not introduced with food, may be looked upon, therefore, as an indication of delayed gastric motility in conjunction with a lowered hydrochloric secretion. These factors are commonly associated when a malignant growth of the stomach exists, and especially in cases where the pyloric region is involved. The finding of lactic acid is, however, not to be considered as pathognomonic of malignancy, though the percentage is certainly high. It has been estimated that fully 85 per cent. of the cases of gastric diseases accompanied by the formation of lactic acid in the stomach is caused by carcinoma.

The quantity of this chemical substance found in foodstuffs is usually small and in almost unappreciable amounts. Oatmeal is without a trace and was therefore selected by Boas as the most suitable substance for testing for lactic acid. The chemical tests are ordinarily carried out only with the fasting stomach contents, and irrespective of the character of food eaten at the previous meal.

The Uffelman Test.—Ten cubic centimeters of a 4 per cent. solution of carbolic acid is diluted with 20 c.c. of distilled water. To this mixture one drop of tincture ferric chlorid is added, which produces a clear amethyst-blue solution. Upon the addition of 5 to 10 drops of filtered gastric contents to this reagent the amethyst color is changed to a greenish yellow when lactic acid is present.

Kelling's Modification.—This is more trustworthy than the Uffelman test, and is to be recommended for routine analysis. A quantity of distilled water in a test tube is faintly colored by the addition of a few drops of a 5 per cent. aqueous solution of ferric chlorid. The solution is now divided into two test tubes. To one, the filtered gastric contents is added drop by drop. If lactic acid is present, a distinctly orange-yellow discoloration makes itself evident as the drops become diffused through the solution. The second test tube is used as a control.

THE MICROSCOPIC EXAMINATION.—The microscopic examination of the stomach contents after a motor meal furnishes valuable information and should always be undertaken as an integral part of the laboratory routine in the course of a gastric analysis. Though the quantity of the residue withdrawn may not be large and food particles may not be discernible, it is nevertheless important that a microscopic examination be instituted in every case. Under normal conditions, the residue obtained from the fasting stomach consists for the most part of a few isolated leukocytes, a limited number of epithelial cells, mostly of the squamous type, the nuclei of cells which have remained after their protoplasm has undergone digestion, a minimal number of bacteria and occasionally certain peculiar spiral bodies, shaped like snail shells, which are derived from the action of the gastric juice on mucus. In the stagnant contents, one finds, in addition, food remnants, abnormal cellular elements, and a gross increase in number and a change in character of the bacteria and fungi present.

Food Remnants.—These present characteristic features under the microscope. Muscle fibres are usually detached and appear as reddish-brown columns upon which fine transverse striations can readily be discerned. Fat globules are rounded in shape and appear highly refractive to light. They take a deep red stain upon the addition of a drop or two of Sudan III. The fatty acid crystals occur as fine needles usually clumped together in the form of bundles or rosettes. The starch cells are made out most readily by their blue discoloration upon adding a drop of weak iodin solution.

Cellular Elements.—Red blood corpuscles are sometimes present when gross evidence of bleeding is lacking. Care must be taken, however, not to confuse red blood-cells or leukocytes with undetached yeast cells.

An increase in the number of pus cells, especially when they occur in clumps, suggests an inflammatory or ulcerative process of the gastric mucosa, if one can be certain that they are not derived extraneously from the esophagus or upper air passages.

In gastritis large numbers of epithelial cells are shed from the gastric mucosa. These cells are mainly of the squamous type, though columnar and goblet cells are not uncommonly encountered.

Tissue remnants may be discovered at times especially in the wash water after gastric lavage in cases of carcinoma or other sloughing processes in the stomach. In such cases they may prove of considerable diagnostic importance.

Bacteria and Other Microorganisms.—The bacteria found in stomach contents have been the object of considerable investigation in recent years. Inflammatory states of the gastric mucosa are accompanied by an increase in the number of bacteria. Staphylococci and streptococci are the predominant organisms found. Their source in the stomach can be traced in many cases to septic conditions in the mouth, gums or tonsils.

Yeast cells belong to the group of vegetable fungi. They are introduced into the stomach commonly with bread and other fermented carbohydrate foods. In stagnant gastric contents the yeast cells multiply rapidly, forming long series of chains. Sarcinae are tetragenous cocci, which form cotton-bale-like packets, quite distinctive in character. They are found in certain foodstuffs and may be discovered occasionally in normal gastric contents, especially where the acidity is high. When a large number of these organisms occur gastric stagnation is certain to be present

The Oppler Boas bacilli, in a vast majority of instances, are found in connection with gastric carcinoma. They are long filamentous organisms, exhibiting small branch-like offshoots, and grow readily only in a non-hydrochloric acid medium. They are stained yellow with iodin and are also Gram positive. As a result of their activity lactic acid is formed from the carbohydrate elements of the food. At times Oppler Boas bacilli are discovered in the microscopic field before lactic acid can be detected chemically and altogether their presence in gastric contents is to be looked upon as of considerable diagnostic importance.

Protozoal organisms are sometimes encountered in the gastric contents, but their presence is to be interpreted as merely incidental to a stagnant condition. Otherwise they possess no pathological significance.

Examination of the Duodenal Contents

For a comprehensive study of the duodenal contents, a previous knowledge of the functional activity of the stomach is required. This should include not only a physical and chemical analysis of the gastric secretion, both at rest and during digestion, but also a record of the motor capacity of the stomach. A reciprocal relationship undoubtedly exists between the functional activity of the stomach and that of the duodenum aud its associated accessory organs and their secretion.

The unmixed duodenal contents obtained from the normal fasting individual is always transparently clear and of a slightly viscid or syrupy consistency. A small amount of bile is usually present, which may not, however, be visible in the gross specimen. If there is no contamination with gastric juice the reaction will be found to be faintly alkaline, especially if the H-Ion concentration method be employed as a test.

The duodenal contents aspirated through a tube is more or less subject to contamination. It consists in the main of pancreatic juice, succus entericus, a varying amount of bile, along with an admixture of mucus and possibly of gastric contents. These constituents vary both qualitatively and quantitatively, and depend upon the type of food ingested, the time withdrawal, and especially upon whether a previous fasting state has existed or not. With the idea in view of promoting uniformity and standardization in the study of the duodenal contents, all tests should be conducted only after a fast of at least 12 hours. The undesirability of admixture with gastric contents is in this manner largely minimized.

METHOD OF OBTAINING DUODENAL CONTENTS FOR ANALYSIS.—As emphasized above, it is best that the patient present himself after a fast of at least 12 hours prior to the introduction of the duodenal tube. Any one of the various types of duodenal tubes may be used, though the authors have found that the one devised and recommended by Jutte of New York is the most applicable for the purpose. The manner of passing the tube into the stomach may be through either the mouth or the nose. According to the authors' experience, the latter method is the least objectionable to the patient. During the passage of the tube through the nose, a resistance is commonly encountered in the posterior nares, which is, however, easily overcome by asking the patient to swallow several times. When the tube has reached the stomach, the initial step includes the removal of all the residual gastric contents, which consists for the most part, of accumulated mucus from the stomach and upper air passages. The patient is now made to lie on his right side and if possible his mind should be distracted by pleasant conversation or the reading of light literature. Approximately 31 inches of the tubing should be fed to the patient. The length required to reach the duodenum depends to a certain extent upon the height of the individual and upon the position occupied by the stomach within the abdominal cavity. It is highly important that the final 6 inches of tubing be introduced quite slowly and with care, so as to prevent coiling within the stomach. When the full measured length of the tube has been reached, the proximal end outside the mouth should be secured by applying adhesive tape to the nose or cheek.

The entire method of procedure should be conducted as nearly as possible along physiological lines, avoiding all extrinsic measures, such as massage movements over the abdomen, as well as the introduction of drugs into the stomach. The drinking of small quantities of tap water, on the other hand, by stimulating gastric peristalsis assists materially in promoting the passage of the tube through the pylorus. An average time of 2 hours is required for the entrance of the tube into the duodenum, under normal conditions. Meyers and McClendon found that an average of three and a half hours was necessary in their cases. According to the authors' observations in over 300 individuals, the minimum time for reaching the duodenum was three quarters of an hour. In cases of gastroptosis, or especially when pylorospasm is present, five or more hours may be required. Failure to reach the duodenum was recorded in about five per cent. of the above cases.

In order to make certain of the entrance of the tube into the duodenum, the following indications may be used as control:

(1) When the tube has found its way into the intestinal tract definite pull or traction may be noted, which is absent when the tube lies in the stomach. Likewise, because of the negative pressure within the duodenum, when the slight withdrawal of the tube is attempted, a certain characteristic tug makes itself felt.

(2) For a similar reason, aspiration of the duodenal contents is not accomplished with the same facility as is the case with stomach contents. Collapse of the rubber tubing is also noted when attempt is made to withdraw the duodenal contents too rapidly.

(3) When the tip of the tube has once entered the duodenum a few

swallows of milk or other colored fluid will not return immediately upon aspiration as is the case with stomach contents.

(4) If the bell of the stethoscope be placed over the epigastric region and a small amount of water is instilled through the tube, a low-pitched gurgling sound is heard when the tube is in the stomach. If, on the other hand, the tube has entered the duodenum, the sound produced by the water is of a higher pitch and repeated splashings will be heard as the water trickles downward through the small bowel.

(5) A few drops of Toepfer's or blue litmus reagent added to stomach contents produces a prompt reddish discoloration, because of the presence of free hydrochloric acid. The neutral or alkaline duodenal secretion remains unchanged on the addition of either reagent.

(6) Whenever conditions permit, fluoroscopic visualization of the tube should be made from time to time, until the tip has been found to have reached the first portion of the duodenum. Fluoroscopic examination, likewise, offers additional information in regard to the type and position of the stomach, the direction of the longitudinal axis and the approximate length of tubing required for entering the duodenum.

Clinical Information Derived from the Examination of Duodenal Contents.—For clinical purposes the examination of duodenal contents may be divided under 3 headings: macroscopic, chemical and microscopic, in a manner similar to those previously described under the head of stomach contents. The macroscopic examination often furnishes important information in the way of an abnormal opacity or discoloration. The presence of a large number of pus cells produces a marked opacity. In the case of pure pus, a milky hue may result. An excessive amount of mucus brings about increased viscosity with stringiness. A reddish or dark brown hue discloses an admixture with blood, usually from an ulcerated surface. Various shades of biliary discoloration have been considered by Einhorn as indicative of certain pathologic states within the gall tract. More recent observations, however, have discredited the importance of these observations. The normal bile color is changed readily by chemical reactions, due mainly to admixture with stomach contents.

Studies of the chemical reactions of the duodenal contents have made rapid advance in recent years. This has been due largely to the introduction of the more exact colorimetric methods, based on H-Ion concentration. In order to test the H-Ion concentration of duodenal contents, brome thymol blue in a 1 per cent. alcoholic solution is used as an indicator. The color range of this indicator is sufficiently sharp to register even slight modifications when compared with standardized color reagents. Myers and McClendon in a series of observations demonstrated that the H-Ion concentration of the duodenal contents in normal individuals, 3 or 4 hours after a meal, varied from slightly acid (3.20) to alkaline (7.2). After a somewhat longer period of fast, Okada and Araia found a distinctly alkaline reaction in the duodenal contents in 8 out of 11 patients examined. In 12 determinations made by these authors during active digestion, an alkaline reaction was present in 7 individuals, an acid reaction in 4 and 1 case proved neutral. In our own series of observations, totaling 35 in all, in which a 15-hour fast preceded the test in each instance, an alkaline reaction was obtained in 30 (7.0 to 8.4). Four were found to be neutral, and 1 was acid.

For ordinary clinical purposes, an approximate estimation of the acidity or the alkalinity of the duodenal contents may be determined by the more simple titration method, employing litmus solution as an indicator.

The microscopic investigation of the duodenal contents serves to supplement the results of the macroscopic and chemical examinations. Under normal conditions, one finds a few scattered leucocytes in the microscopic field, a somewhat larger number of epithelial cells with debris, possibly a few bacteria and an occasional crystal. In the presence of pathological lesions, these elements are usually greatly increased. An appreciable number of pus cells, especially when they appear in clumps and are accompanied by an increase of bacteria, denotes an inflammatory process in the duodenum or the gall. tract. Red blood-cells may be the result of slight trauma from the tube. Large numbers, however, betoken an ulcerative condition in the upper tract. Infestation with protozoal organisms is likewise sometimes encountered in the secretions of the duodenum and gall tract. Lyons, Smithies and the authors particularly have called attention to the occurrence of the vegetative forms of Lamblia or Giardia Intestinalis in aspirated duodenal contents. The flagellates, trichomonas intestinalis and tetramitus meshelii are less frequently found. In 2 cases recently studied by the authors, the larval forms of strongiloides intestinalis were constantly present in great numbers in specimens of the aspirated duodenal contents.

Methods for Determining the Presence of the Pancreatic Enzymes in Duodenal Contents.—*Lipase* (*Steapsin*).—The method of detecting the fat splitting enzyme in duodenal contents is accomplished in the following manner: To 1 drop of neutralized milk, diluted in a few drops of water, is added 3 drops of clear duodenal secretion. A small flake of litmus agar is employed as indicator and the mixture is placed in an incubator at body temperature for one-half hour. In the presence of lipase, the fat content of the milk is converted into fatty acids. The blue litmus is changed red as a result of this reaction.

Buckstein has devised a method based on the same principle which possesses the added advantage of being carried out *in vivo*. An ordinary glass capillary tube is filled with a mixture of olive oil, agar agar and nile blue sulphate, which has previously been heated to moderate temperature. After cooling, a small section of the capillary tube is placed in a perforated, insoluble celluloid capsule to which a silk thread is attached. The patient is now made to swallow the capsule, which finds its way into the duodenum within a short time. At the end of 3 or 4 hours, the thread is withdrawn and the recovered capillary tube is examined for a change of color. A blue color denotes the presence of lipase An estimate of the degrees of lipolytic activity can likewise be obtained from this method by means of millimeter markings placed on the glass capillary tube. The measured length of the color change, expressed in millimeters, represents the approximate amount of lipase present. The existence of a fat splitting enzyme in the gastric juice may serve as a possible source of error in this test, which should not be overlooked. This factor is minimized, however, if the stomach has been kept empty of food by previous fast.

McClure, Wetmore and Reynolds have recently suggested another method for estimating the amount of fat splitting enzyme contained in the pancreatic juice which, though somewhat complicated, is more accurate than the tests previously described. A portion of aspirated duodenal contents is intimately mixed with a similar amount of emulsified cottonseed oil. The mixture after being allowed to incubate for several hours is titrated with a decinormal solution of sodium hydroxide. The degree of acidity determined in this manner serves as an index to the amount of lipase present.

The reagents necessary for this test are as follows: (1) A one-tenth normal sodium hydroxide solution made up in 95 per cent. ethyl alcohol.

(2) A fat emulsion composed of four parts of cottonseed oil, one part of powdered gum acacia and two parts of water prepared in accordance with directions given in the U. S. Pharmacopeia. After preparation, the emulsion should be put in ordinary milk bottles, which are hermetically sealed with patented metal caps. It is then sterilized at 15 lbs. pressure in an autoclave, which insures preservation.

(3) A special molar phosphate mixture solution. This is prepared in the following manner: 53.4425 grams di-sodium phosphate is dissolved in one liter of water. Another solution of 20.4255 grams of acid potassium phosphate is made in half a liter of water. Twenty cubic centimeters of the acid potassium phosphate solution are added to 900 c.c. of the di-sodium phosphate solution which furnishes an H-Ion concentration of exactly 8.4.

Technic.—One cubic centimeter of centrifugalized duodenal contents is diluted in 50 c.c. of the above molar phosphate mixture solution. Nine cubic centimeters each of the fat emulsion and the molar phosphate mixture solution are placed into test tubes which are incubated in a water bath at 40° C. (129.3° F.) for five minutes. One cubic centimeter of the diluted duodenal contents is then added to each tube, the mixture is shaken well, and incubated again for one hour at 40° C. (129.3° F.). Following this, the contents of the tubes are poured at once into small Erlenmeyer flasks of about 150 c.c. capacity, the tubes are rinsed with about 20 c.c. of 95 per cent. ethyl alcohol and the rinsings are added to the flasks (the ethyl alcohol must be neutralized previously with tenth normal sodium hydroxide). Ten drops of a 1 per cent. phenolphthalein solution are added to each flask. The degree of acidity which has developed as a result of the formation of fatty acids is determined by titrating with the tenth normal alcoholic solution of sodium hydroxide. During titration the contents of the flask should be kept at boiling temperature. Control tests should be made with the same solutions, without the addition of duodenal contents. The normal titration figures as determined by McClure for this test varied between 0.9 c.c. to 2.4 c.c. of N/10 sodium hydroxide.

Protease (*Trypsin*).—The presence of trypsin in the duodenal fluid may be detected by adding a small piece of coagulated egg white to the fluid and keeping at body temperature for several hours. At the end of this time, the egg white will have been found completely digested if trypsin is present.

For the quantitative estimation of trypsin, McClure employs soluble casein as the protein material. The underlying principle of this test is as follows: The amount of casein that has failed of digestion, after the mixture has been allowed to stand for several hours in an incubator, is precipitated out of solution upon the addition of metaphosphoric acid. The nitrogen content of this precipitate is estimated on the same principle as that employed for the determination of the non-protein nitrogen of the blood, using an adaptation of the Folin-Wu method. Control tests should be made for the presence of nitrogen in all the reagents employed.

The various reagents are prepared as follows: (1) A 20 per cent. Molar Phosphate Mixture Solution: Add 20 c.c. of a solution of acid potassium phosphate (containing 27.234 grams to the liter) to 980 c.c. of a solution of disodium phosphate (35.628 grams to the liter). This solution gives an H-Ion concentration of 8.4.

(2) A Casein Solution.—One gram of powdered casein is neutralized with 4 c.c. of 1/10 normal sodium hydroxide solution and 100 c.c. of the above 20 per cent. molar phosphate mixture is added. By the aid of heat and vigorous rotation of the flask the casein is made to dissolve completely.

(3) A 25 Per Cent. Metaphosphoric Acid Solution.—The preparation of metaphosphoric acid usually obtained on the market should be fused in a graphite mortar until the white fumes of phosphorus pentoxide begin to appear. The fused material can be cooled conveniently by pouring in a clean pie pan which is floated on cold water. The metaphosphoric acid so fused will maintain its potency as a protein precipitant if kept in tightly stoppered bottles. Solutions should be freshly prepared for each test by triturating the required amount of metaphosphoric acid and water in a mortar.

Technic of the Test.—The duodenal contents are centrifuged until a clear or but slightly turbid supernatant fluid is obtained. The latter is decanted into a clean tube. In a 50 c.c. volumetric flask place 1 c.c. of the duodenal fluid and bring up to the 50 c.c. mark with the 20 per cent. molar phosphate mixture solution.

Into each of a number of test tubes place 9 c.c. of the casein solution. Heat in the water bath at 40° C. (129.3° F.) for five minutes. Then add 1 c.c. of the diluted duodenal contents, mix and incubate in the water bath for 30 minutes at 40° C. Now add 2 c.c. of the freshly prepared 25 per cent. solution of metaphosphoric acid, mix thoroughly and filter. A perfectly clear, colorless filtrate should be obtained. This filtrate is tested for the amount of non-protein nitrogen present, according to an adaptation of the Folin-Wu method, employed in blood chemistry. The digestive activity of protease is expressed in terms of milligrams of nitrogen. Control tests are necessary for accuracy.

The amount of non-protein nitrogen which results from the digestion of the casein the normal duodenal contents varies between 1.46 mg. and 3.57 mg.

Amylase (*Amylopsin*).—The presence of the starch-digesting enzyme in the duodenal fluid is detected by adding a small quantity of boiled starch solution to an equal quantity of neutralized duodenal contents, and incubating from one-half to one hour. Undigested starch assumes a blue color on the addition of iodin. Failure of the blue color to appear discloses complete conversion of the starch into glucose. Erythrodextrin produces a brownish red color reaction.

The quantitative test for amylase employed by McClure follows the principle of the conversion of starch into glucose, and the estimation of the glucose by means of the Folin-Wu method.

The following reagents are needed: (1) a phosphate mixture solution, 20 per cent. molar phosphate mixture of H-Ion concentration 8.4. This is prepared in the same manner as described under the quantitative test for protease.

(2) A starch-phosphate mixture solution. Four grams of soluble starch is dissolved completely in 100 c.c. of hot distilled water. This is cooled and diluted with an equal volume of the 20 per cent. molar phosphate mixture solution.

Procedure for Estimating the Digestive Activity of Analyase.—In a 25 c.c. volumetric flask, place 1 c.c. of the centrifuged duodenal contents, bring up to the 25 c.c. mark with the 20 per cent. molar phosphate mixture and mix thoroughly.

In each of a number of test tubes, place 9 c.c. of the starch-phosphate mixture solution. Heat in the water bath at 40° C. (129.3° F.) for 5 minutes. Then add 1 c.c. of the duodenal contents diluted 1 to 25 as above. Mix and incubate in the water bath at 40° C. (129.3° F.) for 30 minutes. Incubation may be carried out for a period of one hour instead of the 30-minute period, if the starch digestion is found to proceed slowly. The quantity of glucose present in the specimen is estimated according to the principles of the Folin-Wu technic for blood sugar. Control tests are necessary to insure accuracy. The amount of sugar developed by this method in normal duodenal secretion varies between .75 mgm. to 2.07 mgm.

The Einhorn Method for Estimating the Three Pancreatic Enzymes.—Einhorn has suggested a practical method for the quantitative estimation of the pancreatic ferments. All three enzymes may be tested for at the same time by the use of specially prepared agar tubes. The manner of preparation of these tubes is as follows:

(1) Starch Tubes.—Agar powder 2.5 grams; starch 5.0 grams; distilled water up to 100 c.c. Rub the starch in a mortar with sufficient water to make a smooth paste, and then add the remaining water. This mixture is put into a flask, sterilized by boiling, and then drawn by suction into capillary glass tubing (inside diameter 1.5 mm.) which has

been previously warmed. The tubing with its contents is allowed to cool, and is then cut into 3 cm. lengths, and sealed at each end with melted paraffin.

(2) Olive Oil Tubes.—Olive oil 25.0 grams; agar powder 2.0 grams; aqueous solution of Nile blue sulphate (1–2000) sufficient to make 100 c.c. The further procedure is carried out in the same manner as above.

(3) Hemoglobin Tubes.—A specially prepared hemoglobin powder 1.0 gram agar powder 2.0 grams; distilled water sufficient to make 100 c.c. Mix the hemoglobin powder in about 10 c.c. of water until it is smooth, add the agar powder and water, then proceed in the same manner as previously described.

These agar tubes already prepared for use may be obtained on the open market. They should be kept on ice until ready for testing. They retain their efficiency from a month to six weeks, after which the contents become dry and are likely to give inaccurate readings. Before using, Einhorn advises placing the tubes in water for a few minutes (after the paraffin has been removed), especially if the mixtures are not entirely fresh.

Method of Procedure.—One each of the starch, olive oil and hemoglobin agar tubes (with the paraffin scraped off) is placed in a vertical position in a bottle containing the duodenal secretion, and kept at body temperature for 24 hours. A few drops of toluol are added for preservation. At the end of the 24-hour period the agar tubes are removed from the bottle, wiped off, and the various changes, if any, are noted. If trypsin is present the hemoglobin tube will show a transparency extending from the ends. The olive oil tube presents a bluish appearance where the oil has been split into fatty acids by the lipase. The starch agar column should be pushed out of its tube and placed in a weak solution of iodin. All undigested starch will be turned blue. The length of the blue column may be used to determine the approximate degree of starch digestion.

In the same manner, an idea of the approximate degree of digestive activity in the hemoglobin and oil tubes may be obtained by determining the measured lengths of the respective digested columns.

CHAPTER XII

ACUTE AND CHRONIC GASTRITIS. SYPHILIS OF THE STOMACH. LINITIS PLASTICA

By Burrill B. Crohn, M.D.

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Gastritis in General.—Gastritis, acute and chronic, is a diagnosis once commonly in vogue, employed to cover a multitude of errors, and utilized like the term dyspepsia to characterize clinical pictures of indefinite outline and classification. The more exact knowledge of the actual pathological changes in the stomach wall which should accompany the picture of true inflammation, the differentiation of secretory and nervous disturbances and their grouping under a separate head, has tended to restrict the term gastritis to actual pathological changes in the gastric structures. If it depended upon the pathologist at the postmortem table to make the diagnosis of gastritis, it would but rarely be made; nor is today the clinical diagnosis made with any frequency.

The actual changes that characterize " inflammation " as Virchow used the term, " redness, swelling, heat and pain " are practically present in gastritis only as the result of mechanical or chemical trauma. Apart from this, however, we must recognize, at least clinically, milder grades of gastritis, due to dietary indiscretion, decomposing food products, and other noxious substances, in which, while the true pathological picture of inflammation is not attained, yet the congestion of the mucous membrane and the clinical symptoms of a gastric insult can be best classified under the term gastritis. Keeping in mind, therefore, the divergence of the clinical concept, gastritis, from that of the true pathological picture, we may proceed to discuss the subject under consideration.

Acute Gastritis

Acute gastritis may be defined as a pathological change in the mucosa of the stomach, varying from milder stages of congestion, due to the ingestion of bad food or excessive quantities of food, to the more severe grades of actual inflammation due to various toxins, including alcohols and drugs as well as metallic and chemical poisons. In this latter group the pathological picture becomes one of true acute inflammation.

Etiology.—The condition is seen most often in children, particularly infants, but may occur at any age or in any class of life. It has been held that hereditary influences play some rôle; that, in fact, certain families of individuals are predisposed to acute gastric upsets. It is hardly likely that such a condition is hereditary, though a peculiar tendency or idiosyncrasy to certain articles of diet may not infrequently be noted in family groups. In the same way individuals may have certain eccentricities as to diet. Thus a vegetable, fruit or fish which is a perfectly good article of nutrition for the masses may for no accountable reason give rise to marked symptoms in a person who has a weakness toward that particular article.

Seasonal variations may be noted. Thus the summer, particularly the days of dog-heat, is rife with cases of acute gastritis due to decomposed food, shell-fish, spoiled ice-cream, etc., both in adults and in babies. Climate plays no particular rôle. An important factor is the condition of the mouth and teeth as well as the method of and the time taken for the mastication of food. Not without cause did the Army look with disfavor during the War on individuals lacking in molars and therefore devoid of a good chewing surface. Carious teeth, broken teeth and pyorrhea, by giving rise to pain in the jaw and preventing the proper comminution of the food are important predisposing factors. Haste in eating, in the same way, has a definite deleterious effect on the gastric mucosa. Thus a meal properly prepared but eaten in great haste, particularly under nervous tension, may cause much distress and catarrhal gastritis; while the same meal, or even a less digestible meal slowly eaten and properly chewed, with the mind at peace with the world, may be digested with perfect ease.

The preparation of the food is a most important factor, particularly when applied to vegetables. Thus raw starch granules, absolutely indigestible, when properly prepared with sufficient heat either by baking or boiling, becomes one of the most easily digested articles of food. Bad preparation, particularly of the fibrous foods, may well lead to digestive disturbances.

Acute inflammation of the stomach may be excited by the ingestion of food either too hot or too cold. Thus the drinking of very hot water and the tasting of too hot foods as well as indulgence in cold water and iced drinks, the so-called " scourge of the American stomach," may cause inflammatory conditions.

Among the toxic irritants one must consider first alcohol, of which whiskey and gin are the greatest offenders. The ingestion of relatively large amounts, or in these days of prohibition the ingestion of badly distilled or synthetic whiskey, is a very common cause of acute gastritis. It is unlikely that any alcoholic drink containing less than 10 per cent. of alcohol would give rise to inflammatory changes

Chemical toxins taken either accidentally or with suicidal intent, depending upon the concentration and the amount ingested, cause an acute gastritis, varying from a congestion and catarrhal inflammation to an acute inflammatory pseudo-membranous inflammation and necrosis of one or all of the membranes of the stomach wall. Among the chemical agents to be mentioned are:

1. Chemicals having a fixative effect, such as bichlorid of mercury or carbolic acid which cause a membranous necrosis of the superficial layers of the mucosa. The gastric wall has the appearance of a silver gray or a dirty gray covering. The surface of the rugae bearing the brunt of the chemical insult, the mucosa surmounting the fold becomes red and thickened to be followed later by necrosis and an ulceration of the gastric wall.

2. Chemicals having a burning action, such as sulphuric, hydrochloric, nitric acid, etc., producing the pathological picture rather similar to the foregoing, except that there is less membrane formation and more direct maceration and necrosis. Such an injury if survived may be followed by complete regeneration of the mucosa though an atrophic gastritis remains.

3. Chemicals causing a maceration effect such as the alkalies, caustic soda and potash.

4. Milder chemical agents such as oxalic acid, chromic acid, arsenic, phosphorus, etc., which cause a catarrhal or a hemorrhagic inflammation with erosions.

Acute gastritis of bacterial origin is observed as a very rare form under the term " acute phlegmonous gastritis." This is characterized by acute suppurative inflammation diffusing rapidly through the submucosa and mucosa of the stomach causing a huge inflammatory edema with a partial breaking down of the submucosal tissues and pus formation. The offending agent is practically always a streptococcus, rarely a pneumococcus.

Symptomatology.—The first symptoms are those of fatigue, general malaise, aching pains in the back, limbs and head. These are followed soon by the local gastric symptoms. Epigastric discomfort and distension rapidly supervene with nausea and early tendency to vomiting, the latter soon becoming the predominant symptom. The abdominal pain may vary from slight burning sensations and discomfort to severe cramp-like epigastric pain, accompanied by giddiness, faintness, palpitations, cold sweats and collapse. There is profuse sweating and hiccough; profuse salivation is a marked symptom. In a milder case one severe bout of vomiting may terminate the attack; in severer cases vomiting may be repeated and profuse and may be so strenuous as to cause the admixture of traumatic blood. The later vomiting may be that of viscid bile-stained mucus which may continue, accompanied by pain, even after the stomach has been emptied. The amount of vomiting will frequently depend upon the size of the meal ingested and may follow only after the lapse of many hours of gastric stasis. Thus the first vomiting attack may expell food which has lain in the organ for six to twelve or even twenty-four hours after the ingestion of the noxious material. The first effect of the gastritis is to cause a pylorospasm and an inhibition of the emptying power of the stomach.

After manifesting itself at short intervals of twelve hours or longer, the retching and vomiting gradually become less frequent and finally subside. In a milder way the stomach shows the disturbance for several days after the acute attack. Fever in the dietary type, particularly in children, as also in the chemical type and in the phlegmonous type, is not frequent. The tongue is swollen and covered with a thick creamy or a brownish crust and is indented along the margin of the teeth. The breath is offensive and the patient complains of an insipid and bad taste in the mouth. Thirst is almost inevitably present. The abdomen is usually distended and the physical examination may reveal a marked degree of tenderness over the distended viscus. Such tenderness may immediately disappear upon the cleansing of the organ by spontaneous vomiting and the removal of its contents. The bowels are usually costive because of the participation of the intestines in the process or on the other hand may show evidence of a severe diarrhea. An urticarial rash suffusing the skin is frequently observed, particularly in those cases due to fish or shell-fish.

Diagnosis.—The diagnosis of acute gastritis is usually made with facility by analysis of the symptoms, by the nausea and vomiting, and by the history of the ingestion of decomposed food or other toxic or chemical agents. The rapid relief of the symptoms following the vomiting, and the not unusual absence of fever, differentiate it from other upper abdominal inflammatory conditions such as cholelithiasis, perforated gastric or duodenal ulcer and pancreatitis. The attacks of vomiting which accompany migraine headaches may simulate gastritis; the history of repeated previous migraine attacks or the frequent occurrence of migraine in many individuals, particularly women, of one family, will help to differentiate this type of vomiting. The vomiting that accompanies the onset of acute infectious fevers, particularly in children, must also be held in mind. Vomiting as a reflex manifestation of urethral calculus, cholelithiasis, uremia, etc., must be recognized.

Treatment.—From a consideration of the etiological causes, certain simple factors in the prevention of this malady become evident: the **care of the teeth,** the removal of carious dental foci, the insertion of well-fitting and comfortable artificial dental plates, the **proper eating habits,** eating slowly, chewing well, the avoidance of a meal when the mental state is so upset or the emotions so overwrought that abstinence becomes a safer rule than indulgency, and the **avoidance** of **rapid drinking** of excessively hot or cold drinks, particularly in summer. All these factors have much to do with the prevention of repeated acute gastric upsets. The general management of the attack devolves upon the removal of the noxious products, the food decomposition or the chemical agent. **Vomiting** should be **encouraged** or **induced** when not spontaneous. This can most easily be accomplished by asking the patient to drink one or two glasses of plain warm water or that to which a teaspoonful of dry mustard has been added. One to four teaspoonfuls of syrup of ipecac may be helpful. By far the simplest and safest way to induce vomiting is to insert the finger against the posterior wall of the fauces and so originate the vomiting reflex. **Apomorphin** in **one-eighth to one-sixth grain dose** may be used by hypodermic administration, though this method is hardly safe nor to be recommended in cases of acute gastritis due to the ingestion of chemical acids, alkalies, carbolic acid and the like. Where vomiting cannot otherwise be induced, **gastric lavage** becomes indicated and has a very definite and useful sphere of action. Bicarbonate of soda or salt may be added to the lavage water; in cases due to chemical poisons the chemical antidote may be used in the lavage water or may alone be given the patient. Thus the mineral acids call for alkalies such as bicarbonate of soda, milk of magnesia and bismuth. The caustic alkalies should be removed and lavaged. Carbolic acid requires alcohol as an antidote, best given in the form of whiskey, brandy or dilute medicinal alcohol.

Dietary treatment consists in complete abstinence from all solid food; in fact, during an acute attack it is even better to refuse the patient even fluid. Following the attack for one to two days only mild fluids such as milk, tea, gruels, broths and cereals should be given. Too rapid administration of a full diet predisposes to a successive attack. Medicinal treatment is not indicated. Marked loss of appetite or distaste for food following the acute attack may be met by the administration of dilute hydrochloric acid in fifteen minim doses diluted in water and given after meals. The bowels must be kept open freely. This can be accomplished during the attack only by enemas. There should be given after the attack a saline laxative or milk of magnesia, one to two ounces, which will have a similar action and will not cause distress.

Prognosis.—The prognosis in cases due to dietary indiscretion and general food causes is extremely good. The outlook in acute gastritis due to chemicals taken by accident or with suicidal purpose is unfortunately far from good. The course in these cases is that either of immediate collapse with thready pulse, unconsciousness and death, or a later necrosis of the layers of the viscus with perforation and peritonitis. In those fortunate cases where the patient survives the immediate disaster, the pathological picture of an atrophic gastritis with a complete achylia gastrica may supervene. In the milder dietary varieties a chronic gastritis may remain as a sequel of the initial result.

Pathology.—The pathological picture of acute gastritis has already been suggested. One notes marked congestion of the circulatory vessels, infiltration of the interstitial tissue with round cells and leukocytes. Inflammatory edema separates the constituents of the mucosa and in the severe chemical forms membrane formation and necrosis take place. In the milder forms the mucous membrane appears red and congested. The surface of the stomach appears swollen and hyperemic and covered with a thick layer of tenacious mucus. The secretion is diminished and mucoid or mucopurulent, slightly tinged with blood and possessing a fetid and disagreeable odor. The pathological picture in severe cases may extend not only in the mucosa and submucosa but also through the muscular wall and serous covering causing an acute peritonitis. The last is an accompaniment of the form known as acute phlegmonous gastritis.

Chronic Gastritis

Chronic inflammatory changes of the stomach wall may be considered as the end result or the sum total of repeated single insults to the mucosa. Of the non-specific form one recognizes the gastritis due to repeated dietary or thermal traumas, alcohol, chemical poisons, etc. Among the specific forms to be considered are tuberculosis and syphilis of the stomach as well as the rarer form known as linitis plastica.

Non-Specific Chronic Gastritis.—Chronic catarrh of the stomach was the older term under which were grouped the cases of chronic congestion, at times amounting to inflammation, caused by the dietetic errors previously mentioned.

Etiology.—It is unnecessary to recapitulate the etiological factors mentioned under acute gastritis, any one of which sufficiently often repeated might have led to the chronic form of the disease. We must emphasize as predominating factors: (1) The teeth; carious teeth, missing teeth, particularly the molar grinding surfaces and badly fitting artificial plates and bridges; (2). Badly prepared and improperly chosen foods eaten over a long period of time, that is months or years; (3) Excessive use of salt, pepper, paprika and other condiments as it is today so commonly the practice. (4) Food in excessive amounts, gourmandism; (5) Eating of foods too rapidly or while in the state of emotional or nervous excitation; (6) The habit of an irregular dietary.

The life of the busy city, the unnaturally heavy protein breakfast of many of the rural and urban population, the snatched and badly chosen indiscrete lunches, as well as the inordinately prolonged and heavy evening dinner with its heavy desserts, these are factors which cannot fail eventually to make a deleterious impression on all but the man who boasts of a "stomach of iron." Only the exceptional person can stand three heavy protein meals a day, yet many individuals are completely at a loss and have not the faculty of choosing in a restaurant any other than a meat or fish meal.

When taken over a long period many medicinal agents give rise to gastritis, particularly the iodides and bromides; the coal tar products of which aspirin seems to be the main offender; the salicylates; the mercurials, even when not exhibited per oram; the volatile oils, creosote, copaiba and sandal wood oil; many of the narcotics especially the morphin groups.

Alcohol, particularly whiskey, brandy, rum and gin, is notorious for its evil faculty of producing the most classic form of chronic gastritis recognized by clinicians. The gastric catarrh accompanying an alcoholic neuritis or psychosis is a form which in its importance and stubborness to treatment often dominates the clinical picture of these unfortunate maladies. The prolonged use of the stronger alcohols before meals as a so-called stomachic or stimulator of a failing appetite is one of the most vicious forms in which liquor is imbibed. Beer and the milder alcoholic beverages are not usually credited with an ability to arouse chronic gastritis catarrh.

Excessive abuse of coffee and tea are important factors in etiological consideration. The recent observation of such a case in which ten to twelve cups of strong black coffee and as many of strong tea were taken daily has recently given the writer an ample opportunity to observe such an end-result.

Chronic gastritis may also occur as a manifestation secondary to a previous primary disease. Thus with ulcer or carcinoma of the stomach one observes frequently a zone of inflammatory reaction around and about the ulcerating area. Here is observed the truest pathological picture of chronic gastritis, being characterized by round cell infiltration of the interstitial tissue, separation of the alveolae, hyperplasia of the secretory tubules, etc. Yet clinically the primary disease dominates the picture and the chronic gastritis represents a pathological rather than a clinical syndrome.

Chronic gastritis is also described as resulting from myocardial insufficiency and advanced valvular defects. The active factor here, is of course, the passive congestion which involves the stomach as well as the other abdominal organs. While congestion even of an advanced degree is hardly to be classified as an actual inflammation yet the passive congestion which accompanies chronic cardiac and kidney maladies is often similar to that of a primary gastritis. The loss of appetite, the abdominal distress, the vomiting and constant nausea is as rebellious phenomena as any to be met with, particularly where the prolonged administration of drugs such as diuretics, cardiac tonics, etc., have already intensified a deranged digestion. Such an extreme passive congestion for instance as accompanies a chronic pulmonary emphysema with its consequent myocardial weakness may become so extreme as to give rise to duodenal hemorrhage and ulceration. Cirrhosis of the liver, particularly the alcoholic variety, is almost invariably accompanied by a catarrhal gastritis. Arteriosclerosis, occurring as it does most commonly in older persons, is usually associated with a subacidity. In fact, it is generally considered and with good reason, that advancing age and senescence are characterized by lowered acid values in the stomach.

Certain it is that pernicious anemia is accompanied, perhaps caused, by an achylia gastrica which is considered by some to be the end-stage of a progressive chronic gastritis. In some cases the true picture of an atrophy of the mucosa is seen; in others, the achylia is more probably a functional one. The complete absence of acids and ferments may be observed to take place, though the structural and histological elements of the mucosa are preserved in their normal state. While exceptions to the rule are occasionally made, achylia gastrica speaks for pernicious anemia, while retained acid values in the presence of an advanced anemia of unknown origin would tend to point away from primary anemia and would be not inconsistent with a leukemia or a Hodgkin's disease.

The picture of an infectious gastritis due to the ingestion of pathogenic micro-organisms with the food or from diseased teeth has been described and considered by some authors. The writer, however, has so great a confidence in the ability of the acid-bearing mucosa of the stomach to defend itself against the milder forms of bacteria even where the acidity is markedly depressed, that little credence is placed in a form of gastritis, the diagnosis of which is based upon the finding of a few cocci or bacilli in the fasting stomach content.

Symptomatology.—The initial stage of chronic gastritis is rather insidious, extending in many cases over a course of years, it being preceded by a gradual progressive loss of appetite, nausea, distress and discomfort. It may begin as an acute or subacute attack of gastritis which, never subsiding, gradually emerges into the chronic picture of the disease. Pain is not characteristic of the disease. There is abdominal distention with subjective symptoms of discomfort and distention often resulting in a feeling of tightness around the waist, more prominent after meals. There is belching of gas and eructations of bad tasting, sour fluid. Nausea is an almost constant symptom. The patients complain of lassitude, often progressive debility, loss of weight, due both to the gastritis and to the basic underlying cause.

Retching and vomiting are regarded as classical symptoms of chronic gastritis. The characteristic vomiting is that of the early morning or upon arising; a type of vomiting which takes place usually in a fasting stomach, the vomitus consisting of glairy mucus, bile, and if violent, some fresh traumatic blood. The tongue is coated and furred, appetite poor, the bowels generally regular or definitely constipated.

The epigastric discomfort is in the nature of an unpleasant fullness in the region of the stomach an hour or two after meals and is accompanied by a definite bloating of the epigastric area. At this time physical examination would reveal tenderness in this area which may later abate. The discomfort is often associated with an aching or driving sensation between the shoulders or across the small of the back. The milder the meal and the lower the protein content, the less is the distress. The vomitus after the meal is considered to be quite as characteristic and is known as the "vomitus matutinus." Fenwick describes it as "rather abundant in quantity, it presents the appearance of a slimy mass, containing numerous pieces of food which exhibit no signs of digestion. The mucus is uniformly distributed throughout the ejecta and each particle of meat or bread is completely coated with a viscid material. The amount of acid present is inversely proportional to the amount of mucus, free acid being rarely observed. The breath is sickly and offensive and a disgusting taste in the mouth may be experienced upon arising from bed in the morning."

The physical examination will reveal a state of malnutrition and debility, often the signs of an advanced alcoholism in the shape of a neuritis or a cirrhosis of the liver. The epigastrium is usually hypersensitive, at times there may be definite tenderness over the whole area. The splashing characteristic of a dilated atonic stomach may sometimes be observed, particularly where the motility of the stomach has been interferred with. Enlargement of the liver due to fatty degeneration following the abuse of alcohol may be observed as well as the later stage of an ascites, dilated epigastric veins, caput medusae, or other physical signs of a portal obstruction.

Hayem differentiates the hypertrophic form and the interstitial atrophic form of gastritis, ascribing to them respectively a hyperacid gastric secretary curve and an anacid mucoid secretion. The chemical findings in the stomach after the Ewald test meal depend to a great extent upon the normal secretion characteristic of that individual. Thus, the class of individuals who normally show a high acidity would tend to the hyperacid form of gastritis, though probably in the later stages this would eventuate in a subacid or an anacid form. Individuals who normally have subacidity and the smaller percentage of the population who normally have achylia would naturally develop a subacid or atrophic form of the disease. It is unlikely that clear-cut differentiations can be created in the above variations, the degree of acidity present not being sufficient justification for creating a new sub-type. Suffice it to say that the disease is characterized by an increase in mucus, a progressive loss of acid values and in the severer cases, a degenerative process in the mucosa.

Changes in motility of the stomach will depend more or less upon the acidity present. Thus, cases with low acid values and achylia show increased motility and those with higher acid values naturally incline to slightly delayed motility. During an acute exacerbation a definite pylorospasm may be observed, as in one case seen by the author in which following an alcoholic bout a food residue of 24 hours resulted and even on the second day raisins were recovered 12 hours after having been given as a motor test meal.

Diagnosis.—In considering the diagnosis of chronic gastritis one must take into consideration (1) its differentiation from chronic functional disturbances of the stomach and neuroses and (2) carcinoma. The straight-forward types of gastritis due to the continued use of alcohol, medicinal drugs, passive congestion, etc. require to be differentiated from carcinoma. In the latter condition, the x-ray is infallible in at least 95 per cent. of the cases and relieves us of all doubt. Chemically, the differentiation is based upon the finding of marked delayed motility where the pylorus is involved, lactic acid, Boas-Oppler bacilli, and the presence of considerably less mucus than is found with gastritis.

Where long-continued dietary infractions constitute the causative factor, it is difficult to draw a clear-cut line between the picture of the so-called chronic dyspepsia and the chronic gastritis. In the dyspeptic the chemical changes are milder, if noted at all, and the symptoms usually are present in an individual of a neurotic and high-strung temperament. The same dietary insults sufficiently prolonged give rise to the more advanced picture of chronic gastritis.

Treatment.—The first essential of the treatment is the prophylactic avoidance of such causes as give rise to gastritis. This has been fully considered in the discussion of acute gastritis. The institution of a proper diet is one of the essentials. The main object to be kept in view is to **prescribe food** of such a **quality** and of such a **limited quantity** that the enfeebled secretory and motor powers of the stomach are in no way over-taxed. **Excessive fluids** must be **avoided** with meals. Milk, particularly fermented milks, buttermilk, zoolak, etc.; eggs, soups, broths, broiled or baked meats, plain boiled fish, custards and stewed fruits; puréed vegetables should constitute the basis of a proper diet. There is no object in withholding protein foods, since they stimulate acid secretion. Fats and fatty foods in the presence of nausea and vomiting are always wisely restricted.

The use of **saline aperients** and **natural mineral waters** are usually recommended. A stay at French Lick Springs or Saratoga in this country or Carlsbad, Wiesbaden, Marienbad or Vichy in Europe is highly beneficial. The **Bouget mixture** consisting of sodium sulphate 2.0, sodium phosphate 4.0, sodium bicarbonate 8.0, one to four teaspoonfuls in a glass of water before meals, may be endorsed. **Dilute hydrochloric acid, 15 to 30 minim** doses diluted in 2 ounces of water and administered one-half hour after meals, is to be recommended in anacid cases.

For those cases in which vomiting is a troublesome symptom, **gastric lavage** is the ideal method of treatment, either in the good old-fashioned way with a stomach tube and a funnel, the wash water containing bicarbonate of soda which tends to liquify the adherence to the stomach wall, or by means of a continuous lavage as recommended by Rehfuss through a small fractional, flexible rubber tube and tip. Most important is the care of the general health, the association of sufficient exercise and fresh air, vigorous walking, recreative games in the open air, particularly golf. Where the local symptoms are exceedingly annoying, **hot fomentations** to the stomach in the nature of **turpentine stupes** are helpful. It is necessary that the **bowels move freely daily,** the use of the **saline waters** in the morning being the most advantageous way in which this can be assured.

Prognosis.—The course of the disease is a chronic one and when well established may be a progressive one. This holds particularly true as regards the alcoholic cases; the cases due to dietary errors are milder and depending upon the classification of the etiological factors may quite manifestly improve. The prognosis in general is always shadowed by the prognosis of the disease which originates the gastritis. The improvement in the decompensated cardiac lesion or the chronic nephritis, pulmonary emphysema or other lesion being reflected in an improved appetite and diminution of the other gastric disturbing symptoms.

Pathology.—The pathological picture of a chronic gastritis varies from that of a congestion characterized by an increased redness of the mucosa and a secretion of rather thick, ropy, mucoid material to that of a true inflammatory reaction in the wall, resulting from an infiltration of the glandular layer and the interstitial tissue of the mucosa with round cells and plasma cells, congestion of the vascular and lymph spaces, and a gradual increase of the connective tissue elements. Thus, gastritis may be of the earlier or hypertrophic form, frequently associated with a hyperacid secretion and therefore termed by the older clinicians, "gastritis hypertrophicans," or, the cirrhotic element may increase until the picture of an atrophic gastritis prevails. In the hypertrophic form, the irregular enlargement of the longitudinal folds or rugae gives the characteristic picture of the so-called " Etat mamelonné," of the French clinicians. In the advanced forms of gastritis, the superficial mucosa shows advanced mucoid degeneration, the " chief " cells being filled with glassy, transparent mucus. The parallel arrangement of the tubules is disturbed by the inter-tubular cell infiltration and the connective tissue increase.

SYPHILIS OF THE STOMACH

Gastric syphilis is one of the forms of chronic inflammatory gastritis. Up to rather recent years, it was a point of heated dispute, particularly among clinicians, as to whether an anatomical or a pathological picture of gastric lues ever occurred. Until very recently, the author has heard good clinicians still doubting gastric syphilis as an entity. That it does occur in rare cases is presumably no longer a matter of contention, Eusterman having succeeded in collecting from the literature 200 fairly authentic cases and having observed 40 cases in which the serological findings, anatomical findings and the results of therapy establish the clinical and pathological diagnosis beyond the question of a doubt.

Etiology.—The largest percentage of cases confess a primary initial genital lesion; innocent infections account for a smaller percentage of cases. Gastric lues can also occur as a manifestation of congenital syphilis. According to Eusterman the average age of incidence is 35 years. The average duration of the period between the primary lesion and the onset of gastric symptoms is about twelve years, in his experience the shortest period being one year and the longest period 28 years. Thus it is a disease of young life, occuring in the decades when gastric ulcer is commonly present but usually before the patient has entered the cancer period. The disease is much more common in men than in women; cases in infants and young children have not yet been reported.

Symptomatology.—The symptoms will naturally vary with the type of lesion present. Pain, particularly nocturnal pain, is almost invariably present, it occurs as in callous gastric ulcer soon after meals; at first controlled by food, alkalies or liquids, it later in the course of the progress of the disease reacts less and less to these remedial agents. Vomiting soon becomes and remains a prominent symptom, particularly in cases where the pylorus is directly involved or where hour-glass deformity has taken place. In the latter group, with hour-glass stomach, the distress follows ingestion of food which continues causing discomfort, fullness and belching until vomiting supervenes to give relief. If the vomiting is not spontaneous it is usually induced by the patient for the sake of the relief it affords.

Loss of weight is progressive, often extreme, yet again in other cases it is no marked factor. The youth of the average patient, the partial retention of a normal appetite and the absence of the peculiar cachexia of neoplasm are factors which in spite of protracted vomiting tend to preserve the weight of the patient.

The physical examination may reveal tenderness over the pyloric region or in the epigastric triangle. Occasionally a mass may be palpable in the upper abdomen though this is not the rule.

On physical examination there may be observed in the skin, hair, or other viscera or in the nervous system the manifestations of tertiary syphilis including aortitis and enlargement of the liver, spleen, epitrochlear or post-cervical lymph-nodes. In spite of the fact that the neurological forms of the disease, tabes and cerebrospinal lues, are considered rarely, if ever, to accompany visceral luetic lesions, gastric syphilis has been observed in individuals in which irregular pupils, unequal and diminished peripheral reflexes and even the frank signs of developed tabes dorsalis were present.

Diagnosis.—Gastric syphilis is to be suspected whenever a younger individual shows symptoms of rapidly progressing malnutrition, with a well-marked gastric history and particularly with a positive roentgen diagnosis of a deformity of the visceral contour. Protracted vomiting, a pyloric mass of demonstrable defect, an hour-glass deformity in a youthful individual, should always arouse the suspicion of a possible luetic process. The only way to avoid overlooking the specific nature of such a case would be to perform the Wassermann test on all gastric cases which come under observation. While such a procedure is hardly practical under all conditions, the failure to make the necessary serological tests in a suspected case is liable or sure to lead to much humiliation later. Often the true nature of the case is first reported not by the surgeon who removes the mass but by the pathologist who examines histologically the supposedly neoplastic tissue.

It is essential in considering a case with gastric symptoms in the presence of a positive Wassermann or the history or the physical signs of lues, to understand that a benign ulcer of the stomach or duodenum can be present in a patient suffering with a tertiary lues without that ulcer becoming syphilitic. Nor is a benign ulcer affected by the incidence of lues; for the writer has seen a case of benign lesser curvature ulcer in which while under observation and treatment the man developed a chancre and secondary manifestations; the course of the ulcer was unchanged by the presence of lues and unmodified by the specific treatment and positiveness of the serological reaction of the disease. We recognize also that benign ulcer may and does exist in the presence of tabes and even of tabes with gastric crises without that ulcer being in any way of syphilitic origin or construction.

The most constant laboratory finding in gastric lues, apart from the serological test, is the demonstration of the presence of an achylia gastria in the gastric contents. According to Eusterman this achylia is complete in 80 per cent. of the cases, involving a total loss of free acid as well as of ferments. Presumably the process is due in part to direct loss of secreting surface by the pathological process and probably in greater part to the associated chronic gastritis which accompanies the ulcerating lesion. The production of mucus is augmented. Gastric motility in the anacid cases is abnormally hastened; in the cases in which the pylorus is involved in the inflammatory process a delayed motility, such as is seen in any other form of pyloric stenosis, takes place.

One of the more frequent deformities produced in the stomach by the diffuse inflammatory type of the lesion is the "hour-glass" deformity. Such a disfigurement of the viscus is readily demonstrated roentgenographically; it is observed to involve more often the middle or upper section of the body of the organ, rather than, as in benign ulcer deformity, invading the incisura or antrum. The hour-glass deformity of syphilis is more irregular, broader and takes in a greater percentage of the surface area, being in this respect more like that of a malignant deformity. Fortunately, only 1 per cent. of hour-glass disfigurement is due to neoplasm; any such case that does not disappear under the fluoroscopic screen after liberal doses of atropin (differentiation from pseudo or spastic hour-glass) should rightfully be suspected of being luetic in nature. The therapeutic test is in the final analysis the best guide, for a luetic hour-glass does often resolve under specific treatment, as has been well demonstrated by LeWald.

The roentgenographic report of a pyloric deformity or of penetrating ulcer on the curvatures, or of an irregular or broad hour-glass stomach, any of which forms if associated with an achylia gastrica, calls immediately for the suspicion of syphilis and the serological test. Every case of neoplasm should be subjected to a laboratory test for syphilis before subjecting it to operation.

The most important differential diagnosis must be made from carcinoma of the stomach in which disease the same coincident association is seen of a roentgen defect, anacidity and progressive loss of weight and strength. The serological test decides. A provocative Wassermann injection may be necessary before a positive serological test is obtained, and where a fair suspicion of lues is held, should not be omitted.

The course of the disease, if its nature is not recognized, is a progressive though not necessarily a rapid or fatal one. The response to specific treatment is usually most favorable. In the ulcer, in the pyloric form and in the hour-glass deformity type of the malady much depends upon the duration of the illness and the extent to which actual connective tissue scarring has occurred. The complete resolution of the lesion under the specific management can occur, though does not always take place. About 42 per cent. of Eusterman's reported cases regarded themselves as clinically cured, 50 per cent. improved. Many of these on later roentgen examination proved to be anatomically cured as well.

Treatment.—This should be the usual active specific treatment. Weekly intravenous injections of novarsenobenzol over a period of from 6 to 8 weeks are indicated. Throughout the course of these injections intramuscular injections of mercury bichloride or mercury salicylate in oil should be instituted. The continuance of the treatment will depend upon the therapeutic result as evidenced by a change in the roentgenographic outline of the lesion and the relief from symptoms. Where cicatrization of the lesion has occurred recourse is had, after a thorough medical trial, to surgical intervention.

Cases in which the true nature of the lesion is overlooked usually do not respond to medicinal or dietary treatment and eventually fall into the hands of the surgeon for operation. Only thereafter is the true nature of the disease recognized. Pylorectomy or a sub-total gastrectomy may be performed, not always either without a regrettable operative fatality.

Pathology.—Luetic infection of the stomach takes the form of an invasion of the submucosa, the usual histological pathology characteristic of syphilis elsewhere in the body being present, that is, hyperplasia of the connective tissue elements, giant cell formation, hyaline degeneration of the gummatous areas, endarteritis of the blood-vessels of the involved area, etc.

The gross pathological pictures of syphilis of the stomach is usually grouped under three forms which are not clear-cut categories, but which in general cover the anatomical changes which have been observed. The most frequent form is a gummatous mass in the region of and involving the pylorus. This gives a picture both clinically and roentgenologically similar to that of carcinomatous ulceration and obstruction. The second form is that of diffuse gummatous infiltration of the body of the stomach in which the secondary tissue formation may cause an hour-glass deformity of the ventricle of the organ. This is a not uncommon form and has been well demonstrated roentgenologically by LeWald. The third form is that of gummatous ulcer or multiple ulcers appearing on or about the lesser or greater curvatures and simulating in its course that of benign ulcer.

Linitis Plastica

(*Cirrhosis of the Stomach*)

This is one of the rarest forms of gastric disease. It was described by Brinton in 1864 as a rare form of chronic gastritis, characterized by a cirrhotic increase of the connective tissue of the submucosa with a sclerotic contraction of the walls of the organ, causing a general diminution of its cavity. As such, that is, as a chronic inflammatory process, it has been re-described by many authors since. In recent years French writers have made the claim that all such cases are carcinomatous and it would seem that this view may eventually be scientifically established.

Etiology.—Little is known about the etiology of the disease. Alcohol and syphilis have been ascribed as causative factors though probably without good reason. The relationship of carcinoma to linitis plastica remains to be determined. The contention between those who think that all cases of " cirrhosis of the stomach " are of carcinomatous nature and those who believe in a specific sclerosis of the stomach walls is an open one. Many believe with Leith that there is nothing inconsistent or improbable in the supposition of a simple gastric cirrhosis, analogous to a cirrhosis of the liver or kidney or the skin (scleroderma). Atrophy of the mucous membrane is recognized in the form of atrophic gastritis, though here the process involves only the mucous membrane. Similarly one sees the same process involving the serosa of the stomach as part of the picture of a polyserositis, though again the process does not involve the muscularis or the submucosa. Multiple ulcerations of the stomach have been mentioned by Sir Clifford Allbutt as a direct cause of linitis plastica, though probably here we are not dealing with the same clinical picture.

Symptomatology.—The disease runs a long course lasting several years. Sometimes the duration is only that of a few months. The symptoms are practically those of a cancer of the stomach. Pain in the epigastrium is the chief symptom; vomiting, vomiting of watery mucus, later of coffee-ground material or pure blood is frequently present. The hematemesis is due to congestion and at times to ulceration. The tumor may be and usually is palpable in the epigastrium, hard but smooth and but slightly tender. Ascites may be present. Progressive anemia and emaciation advances steadily with rapid loss of weight and strength.

Diagnosis.—The diagnosis is occasionally made before operation or at the bedside. Such a diagnosis must be based upon the finding of an epigastric mass, smoother and not as tender as that of the usual carcinomatous mass, the longer duration of the symptoms and course, and the presence of metastases ascribable to carcinoma.

Today with our roentgenographic facilities the question of linitis plastica arises more frequently. It is not all unusual to see in carcinomata of the stomach such an advanced pathological process that the entire wall of the stomach is infiltrated with neoplastic tissue, so that the lumen is reduced, the walls rigid and tube-like and the motility increased. This might equally well be the x-ray picture of either linitis plastica or of an advanced malignant process. The clinical course helps but little in differentiating the two conditions. The chemical analysis of the stomach offers no help unless the characteristic findings of carcinoma, such as stasis, Oppler-Boas bacilli, lactic acid, etc., are present.

Treatment.—The treatment is that of a chronic gastritis in general. Even where the pylorus is invaded to such an extent as to cause stenosis, it is impossible, because of the tremendous fibroid change throughout the stomach wall, to perform a gastro-enterostomy. Palliative treatment, lavage and finely restricted diet, is the best one can do under these hopeless circumstances.

Pathology.—It is well to quote the admirable description of Leith as illustrative of the pathological anatomy of this condition. "The stomach as a whole is markedly diminished in size, firm, rounded, and elastic. In advanced cases it resembles a thick-walled india-rubber tube, blown out at one end (cardiac). It is increased in weight, and when opened does not collapse. When cut it often gives rise to a creaking sound like cartilage, and there is considerable resistance to the passage of the knife through its walls. The cavity is much reduced; in some cases its capacity falls to little more than two ounces of fluid. The thickening of the wall is greatest in the pyloric region, frequently attaining six to eight times the normal mean, and it may reach the thickness of an inch." The lesion does not pass beyond the pyloric or esophagal apertures. The color of the stomach occasionally is a dull, dead white. On section, all the coats stand out clearly defined. The submucosa layer shows the greatest amount of thickening; next in proportion is the muscular coat. The mucous membrane is rich and mamillated, often surmounted by small projections resembling "gastritis cystica." There is usually evidence of an atrophy and chronic catarrhal inflammatory change of the mucosa. The surface of the stomach contains more mucus than is normal and is occasionally ulcerated. There are diffuse connective tissue bands transversing the mucosa bundles of the muscularis as well as a finer fibrous tissue infiltration. The peritoneal coat is also much thickened. The writer has had the opportunity of seeing two such cases at postmortem in both of which there was no difficulty in demonstrating scirrhus carcinoma of a type in which the epithelial cell nests were so few and the interstitial proliferation so great that if not carefully examined the actual carcinomatous nature of the lesion would have been overlooked. No metastases were demonstrated in these two cases nor have any writers described secondary carcinomatous deposits in the body. Local enlargement of the regional lymph nodes of the stomach have been described.

CHAPTER XIII

ACHYLIA GASTRICA

By Joseph Sailer, M.D.

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Achylia gastrica is a disorder of the secretory mechanism of the stomach, characterized by the cessation of the secretion of hydrochloric acid, and as a secondary disturbance the cessation of the secretion of the ferments of the stomach. In its typical form no hydrochloric acid is secreted at any time, therefore, theoretically, it should be regarded as a permanent condition; rarely, however, does it happen that in these cases, if examined sufficiently often, a temporary secretion of hydrochloric acid fails on some occasion to occur. It is probably, therefore, in all cases more or less intermittent.

The term achylia gastrica has been generally adopted. A variety of other names have been employed none of which appear to have been satisfactory. These usually have the term atrophy associated either with the words stomach or mucous membrane, or with the term catarrh. None of these terms are appropriate, as there is no real atrophy in these conditions, since on histological examination the mucous membrane of the stomach gives a normal picture.

Two forms of achylia gastrica can be recognized, the secondary and the primary. The secondary achylia gastrica is that form in which the failure of the gastric secretion is associated with some other condition which may be regarded as the cause, although very frequently the manner in which such cause produces the effect cannot be clearly explained.

Etiology.—Primary achylia gastrica is the form for which no adequate explanation can be found. It is sometimes ascribed to defective innervation of the gastric glands, to the presence of a catarrhal condition usually associated with displacement of the stomach; but as there is no proof that any of these causes are active, they can all be dismissed. Primary achylia gastrica may give rise to disturbances of nutrition, and it may be associated with other conditions which are in some manner apparently related to the achylia gastrica, either as secondary effects or as clinical manifestations of the unknown underlying cause or causes.

The causes of secondary achylia gastrica are: first, any form of severe chronic anemia, particularly the essential types, such as progressive pernicious anemia and the leukemias; second, carcinoma of the stomach, and also frequently in cachexia due to carcinoma, or any other wasting disease of the body; third, in certain forms of chronic poisoning, such as lead poisoning; fourth, in the stages of exhaustion following prolonged infections, really another manifestation of the cachectic change; fifth, occasionally as a secondary result of other gastro-intestinal disturbances, particularly in sprue. In these conditions, if the cause can be removed, or if the patient recovers, the secretion of the gastric juice returns promptly.

Symptomatology.—Clinical Features.—The patient sometimes complains of a sour taste, indeed this may be the only complaint. It seems to be purely subjective, is rarely relieved temporarily by food, but does not interfere with the appetite. It may cause extreme discomfort. If the tongue is sore it may prevent eating and interfere seriously with nutrition.

The appetite, as has been indicated, is usually good. Occasionally it is diminished, and rarely, as a result of nausea or vomiting, lost completely. Some patients are comfortable as long as they do not exceed a definite number of calories per day. Nutrition if poor nearly always improves rapidly during treatment. About one-fourth of the cases are well nourished throughout the entire course of the disease. Diarrhea is present in less than one-fourth of all cases. This is contrary to the usual statement and the author's figures are taken from a comparatively small number of cases, but it is certainly less common than is ordinarily stated. In some cases, however, the diarrhea is very resistant to treatment. The feces are liquid or soft, occasionally offensive, show little fermentation and the number of movements varies from one to as many as a dozen in twenty-four hours. Dysentery is rare, there is little if any griping, and rarely any pain.

The mental condition is important. Einhorn some years ago called attention to a neurasthenic condition associated with achylia gastrica. The author has observed in several cases a condition which he prefers to call an anxiety neurosis. These patients are constantly preoccupied with their health. They fear some indefinite danger and often distrust themselves, so that they fear to take journeys alone or to assume responsibilities. They are profoundly depressed and often render all the members of their families miserable, but they are not insane, they have no delusions nor hallucinations, do nothing foolish, and their business judgment is unimpaired.

X-ray studies and the fractional test meal indicate that the stomach empties itself in less than the usual time. This may account for the absence of a splash.

Vomiting occasionally occurs and may be so persistent that it endangers the patient's life. It is usually difficult to recognize that the cause of the vomiting is the lack of gastric secretion; but the diagnosis may be suspected if free hydrochloric acid is persistently absent in the vomitus, if the physical signs of conditions that might cause obstruction are absent, if there is no acetone or acetoacetic acid in the urine, and no odor of acetone on the breath, if an x-ray of the stomach is negative, and if alkalies which have usually been administered have failed to improve the condition. The diagnosis can be confirmed only in case the patient responds to treatment for the achylia, and no other cause can be discovered.

Nocturnal pains similar to those observed in cases of duodenal ulcer rarely occur. The author has observed this a few times. They usually awaken the patient about two in the morning, may be appeased by local heat or food but not by alkalies, and are not associated with hunger pain through the day. They are usually relieved or abolished by the acid treatment.

Intense headache of a blinding character associated with dizziness is sometimes associated with achylia gastrica. In the 2 cases that the author has seen, it presented nothing characteristic. The achylia gastrica was discovered during the routine examination, and when treated the headache was relieved.

Illustrative Cases.—Case I.—Male, white, 30. Total acidity 16; free hydrochloric 0; pepsin 0; blood sugar 71.4; 100 grams of glucose given; 30 minutes later, blood sugar 83.3; 60 minutes later, 100; 120 minutes later, 83.3. *Achylia gastrica.*

Case II.—Male, white, 38 years. Fractional examination, total acidity 5 to 8; free hydrochloric 0; pepsin 0. Duodenal contents:—Tryptic and amylolytic ferments strongly positive. *Achylia gastrica, profound neurosis.*

Case III.—Female, white, 45 years. Total acidity 6; free hydrochloric 0; pepsin 0. Duodenal contents:—Tryptic and amylolytic digestion strongly positive. Vibratory conduction absent through bones of legs. Blood picture typical of pernicious anemia.

Case IV.—Female, white, 50 years old. Weight 138 pounds dressed. Diarrhea; apprehensive neurosis; complete achylia gastrica. Ten years later, weight 200 pounds. Mental condition improved.

Diagnosis.—The diagnosis of secondary achylia gastrica is made in the same manner as is that of the primary form, and the symptomatology is that of the causal condition, therefore, it needs no further discussion, except to mention later some of the points in the differential diagnosis.

The diagnosis of the primary form can rarely if ever be made from the history and symptoms. At best it may sometimes be suspected if some of the following symptoms are described by the patient,—sour taste or sore tongue, diarrhea, anxiety about his health, progressive loss of weight; but these may all occur in other conditions. The only definite diagnosis is made by the examination of the gastric contents by the fractional method.

The characteristic finding is persistent absence of free hydrochloric acid, a low total acidity, not over 10 (the statement of Fenwick that it should not exceed 4 to 5 would exclude many otherwise typical cases); no peptic digestion occurs, even if the gastric contents have been properly acidulated, no lactic acid and no Oppler-Boas bacilli. Occult blood is occasionally present as a result of traumatism during aspiration. It is of little significance. Pus is rarely present.

Occasionally yeast plants can be cultivated on acid media. Years ago Jaworski noted that if a hydrochloric acid test meal (if such a term can be used) be given to the patient, the gastric contents withdrawn later contain an active proteolytic ferment. This the author has repeatedly confirmed.

One hundred cubic centimeters of a one-fiftieth normal solution of hydrochloric acid ($^N50$ HCl) is administered through the stomach tube, allowed to remain fifteen minutes in the stomach and the gastric contents then aspirated. This is acidulated to the original strength, one-fiftieth normal, and tested with Mette's tubes.

Jaworski's method is also positive in carcinoma of the stomach, and therefore is of no value in the differential diagnosis.

Although it does not directly aid to establish the diagnosis it is of interest to extract and test also the duodenal contents. This the author has done in 12 cases; in all of these trypsin and amylopsin were present. Bile was also found. As it is supposed that the pancreatic secretion is stimulated by secretin and that this is produced by the action of hydrochloric acid upon the duodenal mucosa, it is obvious that in cases of achylia gastrica some other mechanism is acting. Whatever it is, it is efficient. Trypsin has also been found in the feces.

It may be noted that the pancreatic secretion usually compensates perfectly for the absent gastric secretion, and nutrition may be maintained. So far as the author knows there is no case on record of combined achylia gastrica and pancreatica.

There are no physical signs that definitely indicate the existence of achylia gastrica. Any type of body, the normal, the narrow or the broad may be found. The nutrition is poor in about 60 per cent. of the cases; it is excellent in about 22 per cent. The tongue is normal in more than half the cases; it is often pale and occasionally bright red or shiny or glazed looking. When these changes are present, the patient complains of soreness and tingling and is usually hypersensitive to acids. As these changes also occur in progressive pernicious anemia, in pellagra and in sprue, and are not even frequent in achylia gastrica, they are not essential features of the disease. The examination of the thorax reveals only morbid conditions that have nothing to do with the achylia, or lesions that may cause a secondary achylia gastrica, such as malignant tumors or disturbances of the blood. The examination of the abdomen may reveal active peristalsis, and the absence as a rule of a splash in the stomach, neither very conclusive, and only other conditions such as a tumor or an enlarged liver that might serve to explain the achylia. Tenderness in the region of the gall-bladder is observed in about one-fifth of all cases. A small proportion of these have disease of the gall-bladder. The pulse is usually slightly more frequent than normal.

Differential Diagnosis.—The differential diagnosis must be made from three conditions. Fenwick in 1877 described 4 cases that were probably pernicious anemia. In this disease there is complete loss of gastric secretion and usually a fair degree of nutrition. Pernicious anemia has in addition the peculiar color of the skin, the changes in the blood, the greater degree of prostration, the sensory-motor disturbances. In the early stages of pernicious anemia the author has found the transmission of the vibrations of a tuning fork through the bones of the lower extremities to be diminished or lost. This the author has never found in cases of achylia gastrica. Often the pulse in pernicious anemia is of the Corrigan type and the blood-pressure is higher in the lower extremities than it is in the upper. These observations may serve as additional aids in the differential diagnosis.

In concealed carcinoma with achylia gastrica, the nutrition may be fair and the blood little if at all altered. In these cases, no matter where the carcinoma is situated, the author has found that the sugar tolerance is diminished (Edward's test), and it is not diminished in unaccompanied achylia.

In lead poisoning with achylia, the stippling of the red cells usually suffices to betray the correct diagnosis.

Treatment.—The treatment of achylia gastrica consists in the administration of **hydrochloric acid.** Of the official dilute hydrochloric acid from **1 to 4 c.c.** (**♏xvj to f**ʒi) may be given well diluted before or after each meal. It seems to make little difference which time is selected. Usually the smaller dose suffices. The largest dose (4/0) the author has been able to give only one individual, to a case of pernicious anemia, and it was tolerated without discomfort for several months. Many years ago Abraham Jacoby suggested the administration of hydrochloric acid in milk for children. It is also useful for these cases, enough dilute hydrochloric acid being added to a glass of sweet milk until the reaction to litmus is fairly acid. It is easily taken in this form.

Pepsin may also be administered. There is no proof that it is beneficial, but it is harmless and possibly useful. From 0.5 to 1.0 (grains vii to xv) of a good scale pepsin should be added to the dilute hydrochloric acid, or a mixture can be made of which one teaspoonful represents the dose.

℞	Pepsinal (scale)	0.5	gr. vii ss
	Acidi hydrochlorici diluti	1.0	♏ xvi
	Aquae destillatae q.s.	4.0	fʒi

M. Sig. Three times daily before meals.

If the hydrochloric acid is not tolerated, usually because of a sore tongue, some other acid can be substituted. **Tartaric acid** seems to be the best and the dose is **1.0** (**♏xvj**) three times daily well diluted. It is less efficient.

The diet curiously enough need not be restricted except that all food spoiled (certain cheeses, game) or likely to spoil should be avoided. These patients digest everything well; and usually, if emaciated, gain weight rapidly. A few cases experience discomfort if the amount of food is increased beyond a point of tolerance. In one of the author's cases this was 1,100 calories per day, nevertheless on this amount she gained weight.

The treatment of some of the complications may be noted briefly. The diarrhea requires as a rule only the hydrochloric acid; if this does not check it, aromatic sulphuric acid may be substituted in the same dose. Bismuth and the opiates are rarely useful and often harmful.

The mental condition requires the treatment of the neurasthenia, occupation, moderate exercise, hypernutrition and encouragement. Recovery is, as a rule, very slow, and relapses are common.

Pernicious vomiting is apparently rare, but perhaps it is not always recognized. Acetone is usually found in the urine. The treatment is hydrochloric acid and a diet of buttermilk and the patient must have absolute rest in bed. The author has seen only one case, and the vomiting, which was pernicious in character and had lasted for three weeks, was controlled in forty-eight hours and the patient recovered.

There is no special treatment for the headache nor the nocturnal pains. The former may be controlled by the acid treatment, but there is a tendency to return; the pain usually ceases promptly.

Prognosis.—Prognosis is satisfactory for life, death apparently never occurs as a result of the achylia. Recovery sometimes takes place but it is difficult to estimate its frequency, because remissions are not uncommon and a prolonged remission may be mistaken for permanent restoration of the secretion. Brief remissions are found in nearly all cases if the condition of the gastric contents is carefully watched. They apparently do not indicate improvement. The complications will usually disappear during the treatment of the underlying condition.

Pathology.—The pathology of achylia gastrica is not constant. In the secondary cases various changes may be found in the wall of the stomach, particularly a form of thickening associated with round cell infiltration that may be called chronic gastritis; or there may be a thinning of the wall. Both forms may be found in any form of catarrh of the stomach.

CHAPTER XIV

GASTRIC ULCER, DUODENAL ULCER, EROSIONS, HEMORRHAGE

By Allen A. Jones, M.D.

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GASTRIC ULCER

Synonyms.—Ulcus ventriculi; peptic ulcer; round ulcer of Cruveilhier; digestive ulcer; eroding ulcer; perforating ulcer.

Definition.—Gastric ulcer is usually a single lesion situated most often in the posterior wall of the stomach near the lesser curvature; it is round and punched out in appearance with clean edges. There may be more than one ulcer and the shape and position may vary. A definition is necessarily incomplete and reference to the pathology is needed for further description. The clean cut appearance of the edges is supposed to depend upon its peptic character and the absence of granulation is notable.

Etiology.—This has always been an undetermined question and remains so at the present time. Climate, season, altitude, and soil cannot be said to have a relation here though theoretic arguments might be advanced that they possibly exert a thermic, circulatory, infectious or dietary influence in the causation of ulcer.

Sex.—For many years peptic ulcer was thought to occur much more commonly in young females than in males but the experience of recent years has taught us that gastric ulcer is of more frequent occurrence in males than in females. The individual experience of different clinicians are apt to vary widely in this matter and the diagnosis of gastric ulcer from symptoms alone by the older methods, which relied largely upon the symptoms related by the patient, it today open to serious question and duobtless many mistaken diagnoses were made and statistics unwittingly falsified. For many years, running about twenty to thirty years ago, at a time when the gastric neuroses were probably overestimated, many cases of ulcer in men were mistakenly considered as instances of hyperchlorhydria. It was noticed that nervous, high strung intensive men numerously developed late post-prandial distress or pain in the epigastrium and that they were almost invariably relieved by a diet of milk, eggs and meat, together with the administration of alkalies. Under such a regime their symptoms were controlled but it was noticed that they returned a shorter or a longer time after dietary restriction and the alkaline sedatives were discontinued or disregarded. The classical manifestation of gastric ulcer and that which unequivocally fixed the diagnosis was hematemesis and as that occurred most commonly in the acute bleeding ulcers of young chlorotic women, it was supposed that the disease was most commonly encountered in this sex. At this period in clinical medicine, the finer and more exact methods of analysis of the gastric contents and feces were not in vogue, Einhorn's thread test had not been developed nor were the feces examined for very minute amounts of blood or so-called occult blood. Nor had radiography been elaborated so as to detect gastric ulcers, some of which might now be thus diagnosticated. We now feel sure that many cases of both gastric and duodenal ulcers in men were wrongly called hyperchlorhydria and as this secretory condition was much more frequently encountered in men than in women there was a considerable delay in the awakening of the profession to the fact that ulcer was really more commonly present in men than in women. Furthermore the work of Moynihan, the Mayos, and other surgeons, which has taught us much about gastric and duodenal ulcers, has, since the time above referred to, demonstrated the preponderating frequency of the condition in men. The writer's personal experience has also emphasized the fact that ulcer is more often found in men than in women; the exact percentage difference it is not possible to state. There are apparently many reasons: hurried and injudicious eating and drinking; they have in the past indulged more frequently and liberally in alcoholic drink and in the use of tobacco; their lives are fraught with greater nervous stress and strain; their sleep and rest hours are often insufficient or broken and, indeed, they live for the most part in a manner which puts all their organs on the rack.

AGE.—In reference to the age incidence of ulcer it is seldom found in infancy, childhood or in the adolescent period. It assumes prominence in the early thirties and is found in the succeeding decades to sixty or thereafter. Men present themselves with ulcer most often between thirty and fifty years of age, but in chronic ulcer it is not uncommon to elicit a history of attacks which were very probably due to ulcer, having occurred between twenty and thirty years of age. As there must have been a first-time when a patient felt the ulcer symptoms that time was in the early adult life, a period in which acute ulcer manifestations are often encountered. Irritative gastric symptoms with pain in men in the heyday of life, or well beyond middle life, so apt to depend upon ulcer that exclusion of other causes is called for. It is well known that ulcer symptoms may be stimulated by intragastric diseases in both sexes, but it is well to be as sure as possible that ulcer is or is not present even though extragastric disease may be demonstrable. Deaver's experience in this matter may well be borne in mind as he observed years ago that gastric or duodenal ulcer was apparently secondary to appendicitis in a goodly number of instances. Acute gastric ulcer with hematemesis is met with in young women and it is noticeable that vomiting of copious amounts of blood in these individuals is sometimes the first manifestation of the existence of an ulcer. The writer recalls several instances of this nature, the patient having pursued the usual course of life and not having sought the advice of a physician for relief from stomach symptoms. On the contrary epigastric pain and vomiting may precede the hemorrhage by weeks or months; or indeed macroscopic hemorrhage may be lacking. In older people ulcer symptoms may come into the foreground either because of the development of the ulcer from arteriosclerotic changes, or thrombosis or embolism or because of chronic local peritonitis resulting from attempted perforation and adhesions to the liver or other neighboring structure, or owing to pyloric stenosis. The recurring tendency of ulcer renders it difficult to determine whether a gastric hemorrhage in a man well beyond middle life is the result of a freshly formed ulcer or due to fresh activity in an old area. Experience indicates that fresh ulcers in the later years of life are apt to result from circulatory changes in the gastric wall.

Race.—It appears that ulcer is most commonly encountered in Great Britain, Germany, France and the United States. It has been stated that the Slavonic races are relatively free from ulcer but Stockton regards this as probably doubtful. It has also been held that ulcer occurs with much less frequency among the Japanese and Chinese than among the inhabitants of Western Countries.

Heredity.—*Family Predisposition.*—As there seems to be a family predisposition to vagatonic states and as vagatonia seems linked with a predisposition to pylorospasm, hyperchlorhydria and ulcer it is reasonable to assume an heredity tendency to the development of ulcer near the pylorus. In our experience several branches of the same family have shown this. Two first cousins closely resembling one another have duodenal ulcer, and two brothers of another family have had gastric ulcer. It is, of course, impossible to prove a family predisposition but we are under the impression that it exists and is associated with heredity tendency transmitted along the autonomic nervous system.

Personal Condition.—*Individual Susceptibility.*—The question of immunity to certain infectious processes and of susceptibility to others is here concerned and it seems probable that anaphylactic sensitization to food proteins, infections or other agents may lie hidden in the tissues. Not so speculative, however, is the observation that rest, ease and out-of-door recreation often serve to dissipate ulcer symptoms, pylorospasm and hyperchlorhydria and restore digestive comfort. There can be no reasonable doubt that good general health, with all that it means to muscular, blood and circulatory conditions, militates against the development of ulcer; whereas chlorosis, nervous fatigue and irritability with digestive and other functional upsets predispose to its occurrence.

Social Condition.—While ulcer is seen in the rich and poor it claims many in either social class who lead strenuous lives attended by mental and nervous strain or hardships and who are denied the benefits to health, and to resistance to disease, offered by out-of-door rest and recreation. Coupled with the strenuous life improper foods or habits of eating may bear a part. The worried and jaded banker or business man may suffer from the functional gastric derangements which no doubt precede and predispose to ulcer just as the harassed, unhappy and tired housewife, seamstress, saleswoman, laundress, cook or clerk may do. In these days when the so-called laboring classes have comfortable, sanitary and in many instances luxurious homes, with as good food as the rest of us, there is nothing in their class which *per se* predisposes to ulcer. They, like the vast majority of mankind, constantly neglect health rules and suffer in consequence with those who are supposed to belong to a more fortunate class. Ulcer cases come from the country, from villages, from towns and from cities. They come from the various strata and occupations of life, from those who suffer exposure and hardship and from those who work in warm and well ventilated places. If perchance in the wards of our hospitals we find ulcer in those brought in from an environment of filth, overcrowding and bad ventilation and lighting, it follows that such living conditions predispose to many diseases.

Toxic Agents.—Whatever are the real causative factors of ulcer there are reasons for believing that the injudicious ingestion of alcohol plays a part, notwithstanding, many abstainers are found to have ulcer. Alcohol is a gastric irritant and produces congestion and inflammation of the mucosa. It also interferes with the motor and chemical functions of the stomach. The pyloric region is chiefly affected by alcohol and the functional disturbances are here particularly felt. Its constant overuse furthermore induces hepatic congestion and later hepatitis with a consequent secondary deleterious effect upon gastric circulation and function. The stronger alcohol beverages are especially culpable, but one of the author's patients noticed for years a recurrence of ulcer syptoms after each occasional drinking of champagne. Other instances of the precipitation of ulcer symptoms following the taking of beer or ale have been noticed, therefore the lighter alcoholic beverages have in some people a deleterious effect.

Diet.—While direct trauma resulting from blows upon the upper abdomen may prove a causative factor, the frequent traumatism of the mucosa from dietary errors more commonly initiates the trouble in susceptible individuals, i. e. in those in whom factors of safety are lacking or broken. In those who are prone to pyloric spasm and hyperchlorhydria the repeated ingestion of course, indigestible, irritating food substances may cause a slight erosion which is the starting point of ulcer. But little is known as yet of the relations of food sensitization in the causation of digestive disorders, but while it is speculative there seems good reason from the clinical standpoint for regarding it as probable that certain persons may suffer from local vasomotor and local nutritional disturbances from foods against which they harbor an idiosyncrasy. Some clinical observations have appeared in medical literature in confirmation of such a view. Hyperemia or even an herpetic eruption of the mucous membrane of the stomach may result from such an idiosyncratic food traumatism and pave the way for peptic ulcer. It is well known that some persons suffer from nausea, vomiting or diarrhea if they take certain foods, and repeated experiences with such articles of diet have taught them not to eat them. Shell fish, eggs, cheese, acid fruits, celery, etc. etc., may be the offending foods; their variety is notable.

Worry.—In the foregoing text mention is made of the influence of nervous and mental states upon gastric function but here it is desirable to amplify and emphasize the topic. It is impossible for us to make an accurate estimate of the truly terrible effects of so-called modern civilization upon the functional efficiency of the human brain, nervous system, and vegetative nutritional structures of the body. We have been in the habit of considering the functional state of an organ as a part from its structural condition failing to appreciate the deleterious effects of the former upon the latter. No better illustration of such effect is seen than in the case of the behavior of the arteries and heart under long continued intensive mental strain. The alarming morbidity and mortality from cardiovascular disease is very noticeable in our clinic experience and statistics, and the same influences which cause cordiovascular disease have a damaging effect upon the structure of the other important organs of the body. The manner in which this comes about is more or less speculative, it may be through endocrine disturbance or through otherwise disordered chemistry but it is real and as important as the effect of infections upon organic change. Some day the modus operandi of these influences and their effects will be better understood and measured.

Association with other Diseases.—*Burns and Skin Lesions.*—Duodenal ulcer has been observed to follow extensive surface burns, the explanation being obscure. It may be that infection here plays a part though trophic disturbances may account for the phenomenon.

Debility.—The debility of age may be associated with gastric ulcer owing to circulatory, nutritional and degenerative changes in the mucosa. Focal infection mentioned later may here play a double roll in the causation of both the debility and the ulcer. Anemia in young women has long been accounted as predisposing to ulcer formation but it is probably more often a result than a cause. Cachectic conditions are, however, frequently seen without ulcer development. Indeed it cannot be said that these general states are in any special way etiologically related to ulcer except it be that they are apt to result from the injurious effects of chronic ulcer.

ACUTE INFECTIONS.—There seems to be no authentic evidence that peptic ulcer is caused by the acute infectious diseases although smallpox, typhoid fever and perhaps some other diseases may give rise to ulcer, It is possible, of course, that infectious processes occurring in the course of the eruptive and other fevers may initiate ulcer and that careful history taking may link up the two.

FOCAL INFECTIONS.—During the past few years it has been quite well established that infection arising from foci in the tonsils, about or in the teeth, paranasal sinuses, gall-bladder, appendix, kidney, prostate, tubes, ovaries or elsewhere is an important etiologic factor in both gastric and duodenal ulcers. The research work carried on by Rosenow and others is now very familiar to all students of the subject. It is hard to estimate the influence of other causes which may be conjointly operative with focal infection but it should be taken into consideration. It seems unnecessary now, as the literature is replete with expositions of the causative relation of focal infection to ulcer formation, to enlarge or particularize upon the matter. The importance chiefly attaching to the subject lies in the emphasis it places upon the hematogenous and lymphatic transference of the infection. It is probable that predisposing factors often facilitate the incidence of the local ulcer morbidity. Of some, though probably not equal, importance is the contact infection resulting from swallowing morbid secretions. Streptococci are the most common infective agents in both direct and indirect causation, though staphylococci, pneumococci or other organisms may play a part in some cases. Owing to lack of space the question of focal infection in the etiology of ulcer is necessarily but briefly referred to, but the writer wishes to emphasize its importance especially in reference to the matter of the prevention and treatment of ulcer, the latter being considered in subsequent pages.

TROPHIC DISTURBANCES.—One of the most important considerations in connection with the etiology of ulcer is that which has reference to the local trophic nerve supply of the portion of the stomach affected. This has been for years emphasized by Stockton who has invited attention to the probable herpetic condition of the mucosa preliminary to the development of ulcer. From local trophic nerve disturbances the resistence of the tissues is lowered and they fall more readily a prey to the digestive juice especially if this possesses a high acidity which is accompanied by considerable pyloric spasm. If there occurs a local herpetiform condition of the mucosa it may itself depend upon infection of the nerve elements as herpes is associated with a streptococcus hemorrhagic ganglionitis.

Symptomatology.—CLINICAL HISTORY.—In some cases the early symptoms of ulcer appear in a mild form and so slowly that the patient pays little heed to them and takes, perhaps, various patent dyspepsia remedies. The symptoms may abate to return again. Finally pain becomes so troublesome that the patient either diets and continues to take proprietary medicines or he consults a physician. In other cases the onset of symptoms is more sudden and may even be ushered in by hematemesis. The pain is severe in some instances and is often the symptom which induces the patient to seek a physician's advice. While there is a clinical picture and a symptomatology of gastric ulcer which is almost classic there are also to be observed many quite atypical cases; these will be described and discussed further on. A summarized statement of the clinical history of ulcer is about as follows: The patient usually has a good appetite but notices epigastric distress or pain about two or three hours after eating. With this pain there is a sense of gnawing irritability which may or may not be accompanied by nausea, eructation, regurgitation or vomiting. More or less epigastric tenderness or soreness is present with the pain which lasts, if not relieved by alkalies, food, or by vomiting, until the next meal with and after which it ceases but to return again about two or three hours later. The pain is often present in the night. There is apt to be constipation, loss of weight and in women anemia. The symptoms persist for a few days, weeks or months according to the extent of the lesion and then subside and disappear for varying periods. In some instances they do not return but in most cases they recur and are apt to grow more troublesome and last a longer time. Many recurrences are then experienced over a period covering from five to thirty or more years. As time and experience teach the patient he learns that a diet of milk and eggs and the ingestion of soda give him relief and his history commonly furnishes this information. If hematemesis does not occur and if pyloric stenosis does not develop, the patient goes on for years with a periodic presence and absence of symptoms. He is usually under weight and is apt to present a haggard appearance. If he is discreet he refrains from foods which he knows from experience disagrees with his stomach and thus he may live a tolerably comfortable life. Acid foods and especially uncooked acid fruits, such as grapefruit, aggravate or cause a recurrence of the symptoms and the patients usually find that they are unable to tolerate any but small meals which eventually reduces their caloric intake below their normal needs. During the years of digestive complaint the patient and his relatives, friends and acquaintances are apt to ascribe his symptoms to a variety of causes, nervous indigestion, liver trouble and bowel trouble being most often named. In the ordinary course of events in the case of chronic indurated ulcer pain, or vomiting, or a combination of symptoms causing invalidism, induces the patient to seek competent medical diagnosis and advice. A more detailed discussion of the symptoms of ulcer will now be given.

Pain.—A patient who has a symptom-producing ulcer usually feels quite well or fairly comfortable in the morning before breakfast and complains of no distress until two or three hours after that meal, about 11 A. M. The distress comes on slowly as a rule, but there may be a short period of quite sharp or sickening pain which is felt suddenly after the period of comfort which the patient experiences following the meal. The pain is usually of a peculiar indescribable character, half burning, half gnawing and it seems to reach a peak and then gradually subside for a short time. It is sometimes a sickening distress and is accompanied by a sense of extreme irritability which renders it difficult for the patient, if engaged in business, to concentrate and carry on his work. This pain is more decided and appears at the same time after meals with greater regularity in duodenal than in gastric ulcer. At luncheon time the pain is wretchedly annoying, but after the first portion of the meal has been eaten it lessens and at the end of the meal it has usually disappeared and does not return until four or five o'clock in the afternoon. It is again relieved by eating dinner, but it returns about ten or eleven o'clock in the evening. As the pain appears when the patient thinks his stomach is empty and as it is relieved by food it has been given the familiar name "hunger-pain." As it is temporarily relieved by drinking milk the patient is apt to take it before retiring, but often has a resumption of the distress at one or two o'clock in the night. If no food is then taken the pain gradually lessens and the patient sleeps, awakening in the morning free from discomfort. If a large meal is eaten the pain is delayed in its onset and some cases experience but little, if any, suffering in the forenoon while in the late afternoon and in the night hunger-pain again appears. If acid fruits and coarse indigestible foods, such as raw cabbage with an acid spicy dressing, are taken with an otherwise hearty mixed meal, the pain in the case of the duodenal ulcer, though coming on late after the meal, is apt to last longer than if soothing digestible foods are eaten. In gastric ulcer near or at the pylorus the pain may be indistinguishable from that which occurs in duodenal ulcer. In some instances, however, the pain makes its appearance at an earlier time after eating; it may be only an hour. There are other cases in which it comes only a few minutes after eating though these are the exception. If the ulcer is situated in the pars media or near the cardia distress may supervene early after eating, though the author has studied cases of this nature with symptoms so mild that they were deceptive and radiography alone demonstrated the lesion. The pain is related to peristalsis, pyloric spasm and the acidity of the gastric contents. With a peristaltic wave the pylorus becomes spastic and not until it yields does the pain cease. The severity of the pain varies greatly in different cases; in some it is disabling with a small superficial ulcer, while in others it is not severe though a deep indurated ulcer is present. A persistent severe pain, however, is indicative of a perforating type of ulcer and in many such cases it is the aggravated suffering which finally induces the patient to consent to surgical intervention. Gastric ulcer pain which refuses to yield to diet, rest, and alkalies is usually of the perforative or indurative type. The pain is most commonly felt in the epigastrium, either in the midline or about an inch to the right of it, under the inner portion of the right rectus muscle. With ulcer near the cardia the pain is felt in the left hypochondrium. If there is an associated gastroptosis the pain may be felt in the umbilical region. If the stomach is pushed or drawn to the right the pain is felt further to the right than is usual. In a few cases the pain is felt high in the epigastrium and in some of the author's cases it was substernal; indeed it may be referred well up in the precordial region simulating pain of cardiac or pericardial disease. Sometimes with ulcer on the posterior wall of the stomach near the lesser curvature pain is referred to the left of the spine, about the tenth dorsal vertebra at which point there may be some tenderness. There are cases of ulcer in which the tenderness is sharply localized at all times in the epigastrium a little to the right of the middle line with the pain, boring in character, showing the same localization. Mayo says pain to the right of the midline signifies duodenal ulcer and to the left gastric ulcer. Referred pain is, however, quite common and it also spreads upon the whole gastric region. The cases of continuous severe pain not relieved by ingestion of food and but incompletely and temporarily by alkalies, are apt to have caused a local peritonitis with adhesions to adjacent structures, or extensive infiltration of the wall of the stomach resulting in a deformity known as hour-glass stomach. In a case referred to me a trifid stomach was found at operation by Dr. Joseph Burke at the Sister's Hospital, Buffalo. This apparently resulted from two deforming chronic gastric ulcers at different locations in the stomach. It should be remembered also that unyielding pain may signify a transformation from a benign to a malignant condition at the site of a chronic ulcer. When the ulcer is situated at the pylorus or in the first portion of the duodenum just beyond the pyloric ring continuous, obstinate or oft recurring, resisting pain very temporarily relieved by the ingestion of food signifies pyloric stenosis or peripyloric or periduodenal adhesions.

In the older textbooks and other writings upon gastric ulcer the pain was described as following closely upon meals and as gradually subsiding thereafter until the next meal. In the writings of more recent years the later post-prandial pain is chiefly emphasized. It should not be forgotten that with gastric ulcer the pain may come on very soon after the meal; this is more often found in young women with vomiting and hematemesis, which symptoms are the display of acute ulcer and are usually accompanied also by distinctly localized epigastric tenderness. Wm. J. Mayo says that acute unheralded hematemesis is usually due to some cause other than ulcer. One case, however, may illustrate the early post-prandial pain and the next, similar in that hematemesis occurs, has the onset of pain not earlier than two hours after a meal. Stockton describes cases exemplifying the two types of pain incident in acute bleeding ulcer.

In reference to the motor behavior of the stomach in the presence of gastric or duodenal ulcer with pain Reynolds and McClure observed five types of abnormal motor activity, viz: (1) Exaggerated type of normal peristalsis; (2) irregular peristalsis; (3) antiperistalsis; (4) pylorospasm; and (5) incisura in the greater curvature. They found that "the onset of all types of pain was accompanied by distinct modification in whatever type of motor activities the stomach had previously manifested." The stomach did not empty well during the pain but after its cessation normal type of peristalsis was resumed and it emptied rapidly. With the onset of pain in some cases normal peristalsis gave way to the irregular type or ceased altogether. In others pylorospasm with exaggerated peristalsis developed. Pain was relieved by the administration of 3 grams (46.30 grains) of bicarbonate of soda or 2 grams (30.86 grains) of soda and 1 gram (15.43 grains) of calcium carbonate in 20 c.c. of water and in several observations after such control of pain normal peristalsis was resumed. In one case of incisura with pain soda gave relief, but the incisura persisted.

The pain of ulcer is considered by Hurst to result from inhibition of pyloric relaxation, excessive peristalsis and hypertonus. He found that the application of acid to an ulcer area causes no pain, but he recognizes the frequent association of high acidity, with painful ulcer and attributes the pain to the associated abnormal behavior of peristalsis with increased intragastric tension. He regards such tension as constituting the "adequate stimulus" for the production of gastro-intestinal pain.

Hurst's view as to the cause of pain with gastric and duodenal ulcer does not quite coincide with Reynold's and McClure's observations that decreased peristalsis often accompanied the duration of the pain. Whatever the explanation of the pain it must be admitted that it is most frequently relieved by alkalies.

Vomiting.—Vomiting is not as constant nor common a symptom of gastric ulcer as it was thought to be some thirty years ago. Indeed, most cases presenting themselves for diagnosis do not recite vomiting in the account of their symptoms; or if it is mentioned it has occurred quite infrequently. Severe pain is often complained of without vomiting. There are, however, many exceptions. As nervous individuals are apt to be ulcer victims and as gastric hyperesthesia is a part of such nervousness, vomiting is present in many such cases. Vomiting occurs soon after eating with an irritable stomach or it may be postponed until later when the pain has reached a degree of severity. Sometimes it is a very obstinate and oft repeated symptom and seems to constitute the chief expression of the gastric hyperesthesia associated with ulcer. There are cases in which vomiting does not occur until four or more hours after eating when the stomach is overfilled with highly acid contents and the pylorus is in an unyielding state of spasm; there is usually pain and marked suffering and relief follows copious emesis. A large amount of vomitus does not necessarily signify pyloric stenosis though it suggests it. Sippy has emphasized the fact that ischochymia may entirely disappear under medical treatment and many others, ourselves included, have noted the entire disappearance of retention and vomiting under proper medical regime. Patients with a turgescent condition of the pyloric mucosa combined with an hypertrophied pyloric muscle are apt to vomit quite a large amount of acid chyme during the night. They get along fairly well during the day but there is spastic food retention, the stomach tires late in the day and nocturnal emesis supervenes. When actual pyloric stenosis results from ulcer and gastrectasia develops vomiting occurs when the stomach becomes overdistended and intolerant. Marked differences are noted in cases regarding their gastric tolerance. Some vomit daily or more than once in a day while others are found with the stomach visibly and palpably overdistended and yet they do not complain even of nausea. It was formerly a common experience for us to withdraw large quantities of gastric contents from a dilated distended stomach without obtaining a history of vomiting excepting quite infrequently. The character of the vomited contents will be described under the heading of the examination of the gastric contents.

Hematemesis.—The vomiting of blood which is particularly characteristic of gastric ulcer brings forth a symptom complex somewhat as follows: The patient feels a sudden nausea, faintness, thirst and becomes pale. She may break into a perspiration and she vomits a large amount of dark red blood with or without food, depending upon the time at which the vomiting occurs in relation to the previous meal and upon the motor efficiency of the stomach. It is often found that the patient has risen from bed and repaired to the closet feeling nauseated and experiencing a desire to evacuate the bowels. Not infrequently the patient faints and is found on the bathroom floor or on the floor on the way back to bed. In the author's experience with cases of profuse hematemesis many have taken place in the night. Repetition of the vomiting of blood quite commonly occurs and depends, of course, upon the continuation of the gastrorrhagia which may prove very alarming and, indeed, may so exsanguinate the patient as to render blood transfusion necessary. In some cases the original hemorrhage continues and repeated hematemesis results, while in others the bleeding stops to occur several hours later with renewed vomiting. Gastrorrhagia nearly always induces nausea and vomiting very promptly and a stomach which has previously exhibited great tolerance quickly succumbs to this catastrophe. Bloody stools often follow or may accompany hematemesis and dark feces may be noticed for two or three days thereafter. Hematemesis is frequently seen in young women but some of the most alarming hemorrhages occur in men; indeed in the author's experience more transfusions have been done following gastric hemorrhage in men than in women. There is, however, no rule as repeated alarming hemorrhages are noted in both sexes and at ages ranging from twenty-five to sixty-five or younger or older. In some cases hemorrhage occurs at long intervals, five or ten years for instance, while in others a single hemorrhage takes place in the life history of the disease. Small amounts of blood may be vomited but that type of hematemesis is the exception. If the vomitus fails to show macroscopic blood it may nevertheless be present as microscopic or occult blood and is, strictly speaking, one form of hematemesis.

Perforation.—This is the most dangerous occurrence in ulcer and it takes place in a considerable proportion of the cases. We are now able to predicate perforation in a few cases through radiographic studies and partly by the severe obstinate and boring character of the pain. X-ray shows a crater ulcer. Perforation may be acute or chronic. In the acute the patient is seized with an excruciating epigastric pain which is deep and constant. Vomiting is apt to supervene but does not in all cases Pallor, cold clammy perspiration and a frequent tense pulse are noted. The pain is at first localized, but it is so severe that it spreads over a large area of the upper and central abdomen. Extreme tenderness and rigidity of the abdominal muscles are present. Sometimes there is an urgent desire to evacuate the bowels. The leukocyte count shows an increase of cells and of polynuclears. The blood-pressure is raised in the earlier stages of the calamity and declines again after the pulse has attained a great frequency and has become weak. General peritonitis is soon established, the abdomen is markedly distended and death usually follows in a few days. There are rare exceptions to this fatal termination in those cases fortunate enough to have a very small perforation which is sealed over by plastic lymph owing to the local peritonitis it excites. Prompt surgical measures if resorted to within the first hour or two following the perforation are life saving in the majority of the cases.

Chronic perforation is seen in cases of deep, indurated ulcers, the peritoneal site of which is found adherent to the liver or other structure. The perforation is incomplete in that it is walled off before the peritoneal surface really breaks and it is backed by fibrous tissue which then forms the base of the ulcer and protects peritoneal cavity. Some cases of chronic perforation, however, develop a series of troublesome events. In one of the writer's cases infection traveled from the lesser peritoneal cavity to a region behind the gall-bladder and to the right perirenal tissues and pus was evacuated from both regions. Fever, pain and tenderness marked a long illness with recurrences of septic temperature, leukocytosis, anemia and loss of weight and strength. Several operations were done and finally the patient made a recovery which has lasted to the present time upon duodenal feeding according to Einhorn's method.

The Mayo brothers long since called attention to the deformities and adhesions of and about the stomach, pylorus and duodenum resulting from chronic perforating ulcer. Subphrenic abscess is one of the complications therefrom. They found chronic ulcers perforating in type at some stage of their existence in a large number of cases; and the experiences of many other workers in gastro-enterology corroborate this.

The symptoms of chronic perforating ulcer may subside to return again and as the ulcer is of the crater variety nature seldom succeeds in filling it in and bringing about a permanent cure. Sometimes these crater ulcers bleed but Wm. J. Mayo says bleeding is the exception and not the rule in this type.

Appetite.—Nearly all patients with ulcer have good appetites but they learn to restrict food intake because the suffering is more aggravated and prolonged after each meal. Milk gives relief and patients often take a glass every few hours during the day and night. In some patients appetite fails or there may be anorexia or an indifferent appetite from the beginning. Fear of the pain though it comes several hours after eating serves to interfere with appetite. Anorexia nervosa is sometimes seen, more commonly in women than in men, with ulcer.

Eructation.—Belching is a common symptom with ulcer and it seems to be an expression of motor erethism with hyperesthesia associated with the local irritable ulcer area. Eructation is, however, not often of the hysterical type and it is ordinarily relieved by alkaline gastric sedatives and by milk and eggs.

Regurgitation.—This may occur and prove quite troublesome. Some patients raise the acid gastric contents repeatedly during the digestive period. It smarts and burns the throat and often excites cough. This "heartburn" is so common a symptom of nervous gastric disorder that it is necessary to study its true significance in ulcer cases; that will, of course, be accomplished by the proper investigation of the case. With a greatly relaxed cardiac orifice in these cases there may exist an increased liability to the development of esophageal peptic ulcer—a rare condition.

Anemia.—There is no anemia in most cases. Continuous hemorrhage or repeated small hemorrhages, the blood passing through the intestine, will give rise to a secondary anemia. Profuse hemorrhages as manifested by hematemesis or by large tarry stools induce severe anemia, the red cells falling below two million and the hemoglobin below forty per cent. These are the cases calling sometimes for transfusion. Young women who have chlorosis may have profuse hematemesis which induces great pallor and other symptoms of acute hemorrhagic anemia. These are the cases Hale White regarded as not true gastric ulcer. In both sexes occasional cases of profuse hematemesis have been observed in which on opening the stomach no ulcer could be found.

Loss of Weight.—There is more or less loss of weight with chronic ulcer. Lessened food intake, pain, broken rest, worry over the digestive disorder, possibly anemia and other factors incidental to the disease interfere with nutrition. If pyloric stenosis develops or great deformity of the stomach results vomiting and starvation soon reduce the weight. Some cases finally present marked emaciation. There is no rule, however, as regards loss of weight and many patients with ulcer are apparently quite well nourished provided the later and more serious complications of the process have not developed. The skin and other tissues may be dry if pyloric stenosis and gastrectasia are present and the urine in such cases is apt to be phosphatic and if much soda is taken it may be alkaline.

Constipation.—In most instances the bowels are sluggish, in some marked spastic constipation is present. It results partly from pylorospasm, is a rather constant attendant of pyloric stenosis and food stagnation and is promoted by the diet of milk and eggs the patients resort to for relief. There are exceptions, however, wherein the bowels are regular or loose even in the presence of considerable ischochymia. If chronic appendicitis is associated with ulcer constipation is the more apt to be persistent. If in chronic ulcer the omentum is thickened and colonic adhesions have been formed the constipation is the more obstinate, or, if, as is often seen, gastro-enteroptosis is present colonic stasis is favored.

Headache.—Many years ago Lauder Brunton called attention to a type of frontal headache associated with gastric superacidity and the writer has seen some cases wherein the administration of soda or other alkalies or lavage or vomiting of acid contents relieved such headache occurring in ulcer cases.

THE NERVOUS SYSTEM.—Even in sanguine temperaments the prolonged existence of ulcer induces a low-spirited neurasthenic state. When the disease is present, as it often is, in those of the melancholy type their natural bent is noticeably accentuated. When ulcer is found in the psycho-neurasthenic the symptoms are exaggerated and an anxiety neurosis is apt to become well marked. The symptoms of vagatonia are often outspoken in ulcer cases as illustrated by the hyperchlorhydria, pylorospasm, spastic constipation, vasomotor instability, cold clammy hands and feet, etc. It should not be forgotten that ulcer may coexist with other disorders such as hyperthyroidism, erythema multiform, etc., and a careful general examination should be made in every case for associated diseased conditions.

Gastric Contents.—Great importance has been attached to the discovery of high acid values in the gastric contents of suspected ulcer cases, but it should ever be remembered that many persons have high gastric acidity without ulcer. The majority of ulcer cases have a high gastric acidity which depends upon free and combined hydrochloric acid. The acidity rises rapidly after eating and in one hour is found at 60 or 70 per cent. A single examination may let the matter of acidity rest there but the fractional method of gastric analysis so well popularized by Martin E. Rehfuss has shown that in two hours the figures are often 80, 90 or 100 per cent. and that the acid curve is high at the end of the third hour and is maintained often longer than that. Free HCl in the earlier withdrawals constitutes a lower proportion of the total acidity than the combined acid, but in the later withdrawal after the Ewald test meal the free acid rises in the proportional estimation of the acidity. The amount of contents is not usually large unless pylorospasm unduly retards gastric propulsion or pyloric narrowing exists. The starchy elements of the meal are less well digested than the proteins. Macroscopic blood may be present in the stomach contents. Extreme importance attaches to the question of the presence or absence of blood either in larger or smaller amounts in the contents of ulcer cases. This is emphasized by Alfred Martinet who points out that through the Leon Meunier method of examination for evidence of bleeding in the gastro-intestinal tract most valuable information may be obtained. Watery solutions of the gastric contents render themselves poorly to color reactions with the various tests because the blood is changed to and appears only in the form of hæmatin and it is insoluable in watery solution. It is necessary to introduce into the stomach 200 c.c. of water to which has been added ten drops of ammonia and this is then syphoned out and gives a positive reaction for occult blood by the various tests, Thévenon's especially, for that purpose. After the withdrawal of part of the ammonia solution from the stomach two or three tablespoonfuls of powdered charcoal is given and the black stool when passed is examined for occult blood. Blood in any amount in the gastric or intestinal contents indicates damage to the mucosa somewhere but varicose venules, erosions and congested or inflamed membrane may cause enough hemorrhage to give a positive occult blood reaction. Blood found in the gastro-intestinal tract may originate in the nose, pharynx, mouth, larynx, trachea, bronchi, lungs or œsophagus; so it is necessary to differentiate between such hemorrhage and that which is truly of gastro-intestinal origin. A high hydrochloric acidity and blood in any amount in the stomach argue very strongly in favor of the existence of ulcer, but some cases are observed with normal, moderate or low acidities. In these, evidences of bleeding call for differential diagnosis between ulcer and carcinoma which will be discussed later. If such evidences of bleeding are present with low acidity and the contents of the stomach are otherwise those of a benign condition and some of the important and common symptoms of ulcer above enumerated are present ulcer would be the correct diagnosis in most instances. Radiographic illustration will confirm the diagnosis. Yeast cells or sarcinae in the gastric contents are found as a rule only in case of stagnation, which seldom depends upon atony with ulcer, hypermotility being much more frequently observed.

THE THREAD TEST.—One of the most valuable procedures in the search for ulcer is the Einhorn thread test which is so well and widely known now that its description is unnecessary here. Care should be exercised in the interpretation of thread stains. It is best to insist upon a white diet during the performance of the test or to carry it out with a clean and empty stomach if practicable. Assurance that the Einhorn gold bucket has passed through the pylorus and carried the thread into the duodenum renders the test more valuable as it serves to distinguish the presence of either gastric or duodenal ulcer and assists in determining its approximate location in relation to the pylorus. A well-defined localized bloodstain constitutes valuable evidence of a bleeding area and in conjunction with other symptoms of ulcer affords a rather conclusive confirmatory sign.

RADIOGRAPHIC EXAMINATION.—Acute ulcer may elude radiographic demonstration but the chronic form is usually illustrated by it. There are several changes from the normal gastro-duodenal outline which characterize ulcer. The so-called niche is seen ordinarily on the lesser curvature of the stomach and is sometimes accompanied by incisura of the greater curvature. In some cases a more extensive and ragged filling defect is seen in the prepyloric region, more rarely hour-glass contraction is found as a late result of ulcer cicatrication and adhesion formation. Or a protrusion is seen at some point along the lesser curvature signifying a crater ulcer or one of the perforating type. The pylorus is frequently sharply contracted and gastric hypermotility is often observed. In the duodenum the cap is deformed more or less, being notched, irregular and incomplete. In some cases there is no resemblance to a normal cap, the duodenum in its first part being an irregularly shaped passage. It is difficult to determine sometimes between perigastric and periduodenal adhesions to the gall-bladder or other near-by structures and deformities in contour occasioned by chronic ulcer changes; not infrequently both are present. Peristaltic waves present an interruption in cases showing much scar deformity. If complete pyloric stenosis has taken place the x-ray plate shows a cut off pyloric region with perhaps a faint stream of barium issuing from the stomach; sometimes differentiation from cancer is difficult.

Diagnosis.—In typical cases of gastric ulcer the diagnosis is based upon the irritative nature of the symptoms, the pain and the temporary relief which follows the ingestion of food, the high acidity of the gastric contents which upon analysis are those usually found in a benign condition, the presence of blood or occult blood in the gastric contents either in the fasting or digestive period, the presence of blood or occult blood in the feces, the positive thread test and the characteristic skiagraphic findings. In duodenal ulcer the typical hunger-pain, the usual absence of blood or occult blood from the gastric contents, and its presence in the duodenal contents and in the feces, the positive stain on that portion of the thread which is bile-stained and the deformity of the duodenal cap as revealed in the radiogram are diagnostic.

Difficulties arise in rare cases of latent ulcer and in those cases presenting a typical pain, low gastric acidity, inconclusive thread tests and skiagrams. The diagnosis of gastric ulcer in those who experience pain very soon after food is taken and in whom the pain slowly subsides as the digestive period passes offers a peculiar difficulty as the symptom is contrary to the rule and is one which is to be expected in cases of gastritis with erosions and motor and sensory irritablity. Vomiting soon after meals may occur in such instances. In these as in all cases where ulcer is suspected the search for evidences of some degree of hemorrhage is of prime importance. The gastric contents one hour after an Ewald test-breakfast may appear normal and yield no helpful diagnostic information. Another source of perplexity presents itself in the discovery of achylic gastric contents associated with complaint of hunger-pain. Patients presenting these symptoms and in whose gastric contents either macroscopic or occult blood is discovered, and these findings are associated with x-ray manifestations of ulcer, are sometimes encountered. In the absence of blood in the gastric contents duodenal ulcer may be present and despite the low acidity the pain is of the late post-prandial type.

Sometimes pain is continuous and in no definite relation to meals and in these there is suspicion of a perforating form of ulcer with local peritonitis. Then, there is the hyperchlorhydria which though persistent and accompanied by hyperesthesia and gnawing misery is unassociated with bleeding or radiographic evidence of ulcer. In some patients a heterogenous mass of nervous symptoms accompany well-defined evidences of either gastric or duodenal ulcer.

These and other confusing facts render it necessary to differentiate ulcer from other diseases which may simulate it.

Differential Diagnosis.—Disease of the Appendix.—Gastric symptoms resembling those of ulcer are often caused by chronic appendicitis. These include epigastric pain, eructation, nausea and vomiting. Pyloric spasm also frequently results from appendix disease, sometimes the prepyloric portion of the stomach is thrown into tonic spasm. The reflex effects of chronic appendicitis upon gastric secretion give rise usually to a highly acid contents in which, however, no blood is found. The x-ray picture of the stomach which is the seat of reflex spasm from appendix disease lacks the Haudek's niche or other persistent deformity present in chronic gastric ulcer and the change in the cap which characterizes duodenal ulcer. The appendix is often found visualized and tender on palpation and retains barium beyond the normal time. Frequently it is tortuous and adherent and may be described as retrocecal. It may be unusually long and twisted in strange shapes like a figure eight. It may show kinks and constrictions. Sometimes it retains the opaque substance for many days or weeks if follow-up observations are made. In some cases it is found high in position and its tip may be adherent to the gall-bladder. Repeated examinations of the gastric contents, duodenal contents and feces are necessary in certain instances in order to arrive at a satisfactory differential diagnosis between ulcer and appendicitis. On palpation of the abdomen there is seldom the localized epigastric tenderness which goes with ulcer if the symptoms are due to the appendix, and on the contrary if the appendix is diseased, and there is no ulcer, tenderness is nearly always elicited in the lower right quadrant. In a few cases of pyloric or duodenal ulcer with very marked gastroptosis the tenderness arising therefrom may be felt below the umbilical level.

Hunger-pain may result from the reflex effects of appendicitis and a history of recurrent symptoms over a period of many years may be elicited, therefore the differential diagnosis becomes the more important. Appendicitis and ulcer may exist together but E. Schutz denies any causal relationship between the two. Moynihan, Deaver and others have stressed such a causal relationship.

Gall-bladder Disease.—There is no difficulty in diagnosticating typical attacks of biliary colic but there is nothing typical about some cases of disease of the gall-bladder presenting themselves for study and these cases may simulate ulcer. Gall-bladder disease may follow typhoid fever or other infections; it is more commonly encountered in fat women about middle life than in men; it occasions pain, soreness and tenderness in the right hypochondrium; it is often accompanied by eructation and sometimes by vomiting which fails to afford much relief. The gastric and other symptoms excited by gall-bladder disease are apt to appear irregularly and have no definite time relation to food intake. By the Einhorn and B. B. Vincent Lyon methods of duodenal study it may, in some cases, be determined that gall-bladder disease exists and this is a useful differential procedure. Radiography may visualize gall-stones or give a clue to a thickened or adherent gall-bladder. Palpation may reveal a dilated gall-bladder or a Riedel projecting enlargement of the liver over the cholecyst. A history of attacks of pain and tenderness beneath the liver accompanied by irregular elevations of temperature and by gastric symptoms with, perhaps, a slight leukocytosis, is indicative of some inflammatory affection in the upper right abdominal quadrant, probably cholecystitis.

Nephrolithiasis.—It has probably fallen to the lot of many physicians to meet with an occasional case wherein nephrolithiasis gave rise to referred epigastric pain. In the absence of the signs and symptoms of ulcer urologic study is called for in these cases and it is necessary to exercise great caution in such a study lest small renal pelvic calculi or ureteral stones escape detection. A source of annoying confusion is the occasional presence of definite radiographic evidence of appendix disease with doubtful or indefinite evidence of disease due to urinary calculi.

Pyelitis, ureteritis or perinephritic abscess may give rise to epigastric symptoms which may excite suspicion of possible ulcer. Careful urologic investigation will help to clear up the question. Perinephritic abscess may itself result from slow ulcer perforation but in such cases gastro-duodenal study will have revealed the primary disease. Chills, fever, pain, tenderness, leukocytosis and later tumefaction in the region of the kidney usually attend perinephritic abscess. Dietl's crisis may be so severe as to simulate acute perforation of an ulcer.

Tubercular Peritonitis.—Almost any complex of gastric symptoms may be simulated in some cases of tubercular peritonitis, but the absence of definite manifestations of ulcer as revealed by gastro-duodenal study should be sufficient to put a physician on guard. Sufficient time and observation are necessary in these cases to exclude other diseases and to include the more widespread abdominal pain and tenderness, the irregular manifestations of fever, the probable leukopenia, the presence of peritoneal effusion or of irregularity distributed peritoneal mesenteric or omental thickening and the possible diarrhea of tubercular peritonitis. The von Pirquet test should be employed. Radiographic study with or without the pneumoperitoneal method may show intestinal displacement and be of some diagnostic assistance.

Gastric Carcinoma.—Differential diagnosis presents difficulties in those cases of carcinoma in which free HCl is present and lactic acid is absent. When carcinomatous metamorphosis occurs in an ulcer area and there is yet no notable food stagnation, nor other evidence of cancer, time and study are necessary to arrive at a satisfactory differential conclusion. The Wolf-Junghans method may be useful here. Radiographic study may be of service. A most helpful observation employed by Marcinet should here be mentioned. He emphasizes the importance of determining whether or not blood in the feces is derived from the higher or lowest segment of the gastro-intestinal tract, i. e., whether it appears as hematin or water soluble blood. Then, "If in a patient with a doubtful diagnosis of ulcer or neoplasm of the body of the stomach, chemical investigation reveals the presence of blood, the patient should be kept in bed for a week and a test for blood carried out at regular intervals. In the event of ulcer the blood will generally disappear before the end of the week. In the event of cancer the blood nearly always persists throughout the rest period."

It is very important to differentiate between gastric ulcer and carcinoma and the literature is replete with reference to the subject; but careful as some studies may be it, nevertheless, becomes necessary to perform gastro-enterostomy with ulcer resection in order to make a positive diagnosis. In cases of primary carcinoma of the stomach beginning at or near the pylorus the difficulty is usually not so great unless early ulceration of the neoplasm and bleeding occurs. These cases usually fail to show the hyperchlorhydria so common with ulcer, there is no history of recurrent attacks with ulcer symptoms; on the contrary it is that of a slow onset of a primary gastric affection usually in a person beyond middle life. The symptoms occupy all of the digestive period and food stagnation with vomiting develops in a comparatively short time. The patient with ulcer usually improves under proper medical treatment, whereas the patient with cancer does not. In most cases loss of weight is progressive and anemia becomes manifest. If the fasting gastric residue is examined increased albumen and perhaps carcinoma cells are found. Repeated examination of the gastric contents following test-meals shows a progressive failure of the secretion of HCl and pepsin and the appearance of Opper-Boas bacilli and lactic acid. In an ulcer case without antacid medication examinations of the gastric contents made day by day will nearly always show a persistent hyperchlorhydria or, if the case be one of ulcer with low acidity when first examined, lavage and subsequent analysis often show a rising restored acidity.

If then the history is carefully taken and considered and careful study is made for a couple of weeks in doubtful cases, it is usually possible to differentiate between ulcer and cancer before tumor is palpable in the latter. Mistakes are easily made by clinicians if opinion is based upon only one examination of the stomach contents.

Pancreatic Disease.—There are a few diseases of the pancreas which are likely to be confused with ulcer. Hemorrhage into the pancreas or acute hemorrhagic pancreatitis may simulate acute perforation of an ulcer. Both are apt to be attended by very severe epigastric pain, vomiting, frequent small tense pulse and abdominal rigidity with extreme tenderness in the early stages and an evident degree of shock. With acute pancreatic disease a history of ulcer will probably not be obtainable. Both conditions induce an acute abdomen and it is wrong to delay surgical measures long while speculating upon the diagnosis.

Chronic pancreatitis is occasionally fraught with recurring epigastric pain and vomiting such as may occur with ulcer. A history of gall-bladder disease and the possible presence of glycosuria are to be taken into account. Coupled with these may be loss of weight and diarrhea with stools containing excess of undigested fat, meat fibres and starches.

Rare cases of pancreatic calculi may also be thought of in connection with pancreatic disease. It may be quite impossible to arrive at a conclusive diagnosis of this calculous affection but ulcer may usually be affirmed or excluded by careful study.

Embolism or Thrombosis of the Mesenteric Vessels.—In this catastrophe we have another clinical condition which may resemble acute perforation of an ulcer and which calls for surgical intervention as no time may be wisely allowed for prolonged differential study which may be quickly prosecuted by the surgeon.

Gastralgia.—Though extreme caution should be observed in making a diagnosis of gastralgia there are a few instances which defy any other conclusion. It is necessary to exclude all other possible causes of epigastric pain and one of those is syphilis of the spinal cord or nerves.

Syphilis.—Gastric manifestations of syphilis have been recorded by Einhorn, Stockton, the author and many others. It may appear in the form of gumma, gastritis or ulceration; or it may assume the form of severe gastralgia as in tabes dorsalis or syphilitic disease of spinal nerve roots and trunks. A careful history taking and a probable positive Wassermann may serve to clear away diagnostic doubt.

Epigastric Hernia.—Cases of epigastric hernia have been observed in which the symptoms closely resembled those of gastric or duodenal ulcer. In an interesting and valuable study of this subject Meyer and Ivy call attention to the references of Kelling, Strauss, Ury, Cohnheim, Soper and Hall regarding the relation of epigastric hernia to the etiology and symptomatology of ulcer. They report a case which came to operation and in which "hunger-like" pains were present. No ulcer was found but a small epigastric hernia with omentocele was encountered; and two years have elapsed since its cure by operation without recurrence of the symptoms. By operation they produced experimental epigastric hernia in 13 dogs and demonstrated a "tugging" action by the gastric omentum which was included in the hernia upon the greater curvature of the stomach in the prepyloric part. No ulcers developed in these dogs. A pouched condition of the stomach was induced. They conclude that gastric ulcer is a coincidence, if present at all, in cases of epigastric hernia and that the latter condition is not a proved cause of the former though as Soper believes it may interfere with the healing of an ulcer. In the differential diagnosis of ulcer it should be borne in mind that epigastric hernia may cause symptoms closely analagous to those of ulcer.

*Radiographic Observation**.—The x-ray examination of the stomach with special reference to gastric ulcer is best accomplished by using both the direct and indirect method.

The direct method consists in demonstrating on films or plates the stomach after suitable opaque meal has been given. The indirect method is accomplished by dluoroscopy and considering "symptom-complex" of the continental investigators.

The x-ray offers valuable assistance in the diagnosis of gastric ulcer although the method is not sufficiently well developed to be relied upon without considering the clinical aspects of the disease. Various observers have placed the value as high as from 75 to 85 per cent. in proven cases and some higher. The diagnosis of gastric ulcer depends upon finding a filling defect and also considering the manner in which the stomach functions during the x-ray examination.

The location of gastric ulcers plays a large part in demonstrating them radiographically. When the filling defect or an ulcer is located on the anterior surface of the stomach or on the anterior surface of the lesser or greater curvature they are usually demonstrable. Regardless of where the ulcer is located the function of the stomach is interfered with or materially changed from the normal. In this condition we have an irritation from the ulcer which causes spasticity, hypermotility and often a shutting off of the pyloric sphincter to such a degree that practically none of the Barium passes out of the stomach. There are however, intervals when the sphincter relaxes so that the meal passes on; therefore, in gastric ulcer we uually find a six-hour residue of the opaque meal.

In many known cases of ulcer these functional disturbances of the stomach are so persistent that we feel that these signs are as important in concluding a positive diagnosis for ulcer without a filling defect. However, the stomach can be so disturbed from reflex causes other than gastric ulcer; so that we must take this point into serious consideration, although the number of cases in which it occurs are few.

Those gastric ulcers which are located near the pylorus frequently give us obstructive signs which aid in making our diagnosis.

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*This radiographic study was kindly supplied by Dr. Edward Charles Koenig, Associate Professor in Radiology, University of Buffalo, and attending Rœntgenologist, Buffalo General Hospital, Buffalo, N. Y.

When the ulcer has been definitely demonstrated x-ray furnishes us a means of deciding the necessity of surgical interference or the progress of the ulcer whether healed or recurring.

Treatment.—The treatment of gastric ulcer should be considered under two headings: First, the treatment of acute ulcer and second, that of chronic ulcer.

ACUTE ULCER.—The first and most important measure is **rest in bed.** If hematemesis has occurred or if bleeding is taking place through the intestinal tract, **starvation** until hemorrhage ceases is sound practice. As a rule bleeding stops within a week after rest is enforced. **Cold compresses** or a **Leiter coil** or a **light icebag** should be applied to the epigastrium for three hours at a time and removed for one hour. If the patient is comfortable sleep need not be disturbed in the night. Thirst should be allayed by **cracked ice** in small amounts per orem and fluids may be supplied per rectum by the drop method (the well-known Murphy drip). One pint of plain water, normal salt solution, a weak **soda solution** or **ten per cent. glucose solution** should thus be given two or three times each twenty-four hours. If hemorrhage has been so severe and copious as to reduce the red blood-cell count below two million and the hemoglobin below forty per cent. **blood transfusion** should be done if possible. In many cases life has been saved or convalescence greatly hastened by this procedure. In the writer's experience it has proven so valuable that he commends it enthusiastically as it accomplishes a larger purpose than the mere hypodermic or intravenous use of coagulose, thrombo-plastin or coagulen, which have frequently been employed in the hope that they would check the hemorrhage and possibly prevent its recurrence. The time-honored hypodermic of **morphine** and **atropin** allays fear and aids in checking hemorrhage. Pantopon is preferable to morphine as the latter induces pyloric spasm. Papaverine would be preferable to either.

After hemorrhage has ceased the plan of treatment laid down by von Leube is most excellent and efficient. He directed that the patient's abdomen should be washed with warm water and soap, bathed with warm sterile water, then with alcohol and finally with 1-5000 bichloride of mercury solution. Then a linen cloth covered with five or ten per cent. boracic acid ointment should be placed upon the abdomen and over that poultices should be applied every 15 minutes day and night. Daily the boric ointment should be washed off, the skin bathed as directed above and fresh ointment applied and the poulticing continued. It is seldom possible to carry out von Leube's directions so strictly and literally. It is unnecessary to apply the poultices so frequently as they may be kept hot by applying a thin rubber tissue, dry compresses and an electric thermopad over them. The writer has for years instructed the nurse to make light poultices of one-third flaxseed meal and two-thirds bran. It is not necessary to rewash the abdomen more often than once daily.

The skin soon becomes mottled under the continued application of heat and later a pigmentation occurs which is slow in disappearing but dermatitis is rare.

The effects of this plan of poulticing are these: the pain, pyloric spasm and gastro hypersecretion are promptly allayed and the healing of the ulcer is accelerated. The von Leube diet consists of boiled milk five times a day during the period of poulticing, meat solution and softened zweibach. The following week add soups, sago or rice thoroughly boiled in milk, white of egg, raw or soft boiled eggs, calf's brain and boiled chicken. Other tender meats are gradually added and after the fifth week a diet of health is resumed.

The Lenhartz Treatment.—In contrast to the von Leube diet and the restricted diets prescribed by most clinicians early in ulcer cases the Lenhartz plan boldly attempts to maintain the nutrition of the patient at a high level by beginning high proteid feeding very soon after the hemorrhage has occurred. The method carried out is as follows: Complete rest in bed is enjoned for four weeks and an icebag is applied over the stomach for a fortnight. Two hundred to 300 c.c. of iced milk in very small quantities at a time and 2 to 4 raw beaten eggs are given the first day. The eggs may be flavored and sweetened slightly and are served very cold. One hundred cubic centimeters of milk and one egg are added daily for the first week and the amounts then reached are maintained for the second week. On the sixth day of treatment 35 grams (540.05 grains) of raw chopped meat is given.

In the Lenhartz treatment bismuth subnitrate is given from the beginning in 2 gram (30.86 grains) doses three times daily and its administration is continued for 10 days. The amount of food given is increased daily until by the end of two weeks the caloric value of the diet is approximately 3000, and the foods given are milk, eggs, sugar, ground rice, zweibach, raw beef, and ham and butter.

The Einhorn Duodenal Feeding.—This is an excellent method of feeding in acute ulcer, and in some cases of chronic ulcer, and one which insures physiologic rest to the stomach and yet affords the patient a fair caloric feeding; it has been carried out by W. Gerry Morgan, Stockton, Friedenwald, the author and many others. The method is now so well known that its detailed description is unnecessary here.

The advantages are that the ulcer is given an opportunity to heal, pain usually disappears on the second or third day after duodenal feeding is begun, thirst is allayed and the unpleasant features of rectal alimentation are avoided. Edsall long since showed how meagre is the amount of nourishment absorbed by rectal feeding. The patients who tolerate duodenal feeding will soon grow accustomed to the presence of the small rubber tubing in the mouth and throat, salivation and reflex nausea disappear and sleep is fairly comfortable. It is sometimes possible to continue the method for three weeks though fresh tubing is advisable after the first 10 days. Indeed, it is often well to withdraw the tube and capsule for the night when it is easily swallowed and passed quickly into the duodenum. It relieves the patient and affords an opportunity for more adequate cleaning of the instrument. Unhappily there are many nervous hyperesthetic individuals who cannot retain the tube even long enough to allow the perforated capsule to pass the pylorus. In others there is a marked delay in the passage of the tube into the duodenum either because it is twisted or knotted or is obstructed by pyloric spasm or partial stenosis. B. B. Vincent Lyon's sinker may be used to lead the capsule and tube through the pylorus. The erethism, worry, excitement and misery manifested by some patients are so great that the method is necessarily abandoned.

The Sippy Treatment.—The most systematic and rigid plan of medical treatment for gastric and duodenal ulcers has been conscientiously, carefully, and thoughtfully worked out by Bertram W. Sippy. His method has attracted much well-merited attention and is now familiar to many physicians but, as is the case with forms of treatment devised by others in the past, wrongful and damaging omissions or modifications are frequently made by those who suppose or feel that they are employing the Sippy treatment. It is this free and unwarranted alteration of the treatment, which is plausibly called a modification and which is often so drastic that the true method is unrecognizable in the quasi-plan adopted, that leads to failure and disappointment and casts unfair discredit upon a truly admirable treatment. The author considers it only safe and fair for Dr. Sippy and for the patients submitted to his plan to give its details in his own words and to urge those who adopt this method to adhere closely to his instructions. Notwithstanding the fact that a few cases of ulcer are encountered in which the gastric acidity is low, Sippy contends that in the vast majority the ulcer is prevented from healing by the corrosive action of the acid gastric juice and that such action must be prevented by the neutralization of acidity through the free and frequently repeated administration of alkalies.

"Three ounces of a mixture of equal parts of milk and cream are given every hour from 7 A. M. to 7 P. M. After one or two days a soft egg with cracker or bread and butter may be added to one of the forenoon feedings, three ounces of a cereal, such as well cooked rice, oatmeal or farina may be added to one of the afternoon feedings. The cereal is measured after it is prepared. Gradually egg and cereal are added until at the end of the first week the patient usually is taking each day three ounces of the milk and cream mixture every hour from 7 A. M. until 7 P. M., and in addition two or three soft eggs, one at a time, and 6 to 9 ounces of a cereal, 3 ounces at one feeding. The cereal and egg are given alternatingly, and at the time and in addition to the 3 ounce mixture of milk and cream. Custards, cream, soups, vegetable purees, and other soft palatable foods may be substituted now and then for the milk and cream feedings. Jellies and marmalades may be gradually added if desired. The patient should be weighed. If desired, a sufficient quantity of food may be given to cause a gain of two or three pounds each week. Control of the free hydrochloric acidity should be maintained from the beginning.

"In cases of peptic ulcer unassociated with stagnation of food and secretion, the free acidity is usually controlled by feeding every hour in the manner outlined, and giving a powder containing 10 grains each of heavy calcined magnesia and sodium bicarbonate, alternating with a powder containing 10 grains of calcium carbonate and 30 grains of sodium bicarbonate, midway between feedings. In addition, the powders should be given every half hour after the last feeding for four or five doses, until the stomach contains no food as is occasionally determined by the use of the tube. Calcined magnesia and calcium carbonate have respectively about four and two and one-half times the neurtalizing power of sodium bicarbonate."

As the Sippy treatment is the outstanding and most systematic plan of medical treatment of ulcer ever devised it is deemed wise to emphasize its essential details. The purpose of the treatment is the prevention of the corrosive action of hydrochloric acidity upon the ulcer area and thus allow the lesion or lesions to heal. It is axiomatic that healing will occur if the acidity is adequately neutralized, therefore, exact instructions are given for the administration of the alkaline powders. It is evident that Sippy feels we are apt to err in giving too little neutralizing powder than too much, therefore, he instructs that a powder composed of 10 grains each of the heavy calcined magnesia, and a sodium bicarbonate should be given every two hours midway between feedings, alternating with a powder composed of 10 grains of calcium carbonate and 30 grains of sodium bicarbonate. As the milk and cream mixture is to be given every hour from 7 A. M. until 7 P. M., the powders are given alternately, i. e., a neutralizing powder ever hour until 7:30 P. M., and then every half hour until four or five doses have been given after the last feediing in the evening. This is done for the purpose of thoroughly neutralizing the free acidity until the stomach has emptied itself of all food. The stomach tube should be used often enough to ascertain whether or not the food remains have passed on and also the degree of acidity present. It will be noticed in the quotations that eggs, bread and cereal are added to the diet after the first day or two of a strickly milk and cream diet and that a few days later custards, cream soups, vegetable purees and other soft foods are added to the diet, which at the end of a week of treatment may be adequate to allow a gain in weight of from 2 to 3 pounds a week. The instructions provide that the calcium carbonate and soda bicarbonate powders are to be given more frequently than the magnesia and soda powders if the magnesia proves too laxative, but it is stated that a moderate looseness of the bowels is desirable if it gives rise to no discomfort.

Sippy directs that the gastric contents should be withdrawn and examined in a routine way on two afternoons and three evenings of each week unless there exists some contraindication to the passage of the stomach tube. He says the evening aspirations should be half an hour after taking the last powder. He has found that when the free acidity is controlled late in the afternoon just before a feeding or a powder it is likely to be under control at other times during the day and early evening. As cases differ the acidity in some is not so easily controlled as it is in others and when necessary the dose of alkali is gradually increased by addition to 5 grains of calcium carbonate to each powder until the desired result is obtained. It is pointed out that "the most favorable conditions for the healing of peptic ulcer are maintained when the aspirated stomach contents show absence of free hydrochloric acid during the entire time that food and its accompanying secretion are present in the stomach and all excessive night secretion is controlled." In the Sippy treatment at the beginning of the fourth week the routine is changed to three small meals a day with a continuation of the milk and cream every hour and the powders between as usual. The total amount of food allowed at any one meal is not to exceed 10 or 15 ounces, measured after it is prepared for eating, but water, tea, coffee and thin soups are not estimated in this amount. The small meals consist of vegetable purees, potatoes, cooked fruits, ordinary desserts, and after a few weeks raw fruits are allowed. Meats, with the exception of bacon, and meat broths are not given until after the termination of the fourth week as they interfere with analyses of the gastric contents and stools. After the establishment of the small meals in the plan of treatment Sippy says the following plan has been thoroughly tested by experience:

"After the small breakfast, a powder should be taken every half hour for two or three doses, depending on the size of the breakfast. One-half hour after the last powder, 3 ounces (90 c.c.) of the mixture, equal parts milk and cream should be taken, the next half hour a powder, and in that manner the milk and cream mixture and the powders should be taken alternately on the half hour until the noon meal. After the small noon meal, a powder should be taken every half hour for three doses, then the milk and cream mixture alternated with the powder every half hour. To lessen the number of powders required after the evening meal the supper should not exceed 8 or 10 ounces. (240 to 300 c. c.). After supper a powder should be taken every half hour for four or five hours. In the absence of pyloric obstruction, if the tube shows that the stomach is practically empty one-half hour after the last powder, the evening aspiration performed three evenings a week to determine whether the acidity has been controlled may be discontinued at the end of the fourth week. Upon resuming normal activity or work, the patient continues the same management. He eats the three small meals at home or wherever it may be convenient. Milk and cream mixed, equal parts, is taken with him to his place of business. A thermos bottle is a desirable container. From this supply a flat flask may be filled and carried in the pocket. A similar flask may contain water with which the powders may be taken. It is often deemed less trouble to take the powders with a swallow of milk.

"Now and then if desired the patient may take an ordinary full meal, or for two or three days at a time, he may take three ordinary full meals daily. After a full meal, one powder each half hour for six doses or two powders at the end of each hour for three doses should be taken. This enables the patient to attend dinners and occassionally break the monotony of the management should it be found irksome. Relatively few seem inclined to take advantage of the privilege. The acidity is not as accurately controlled and the best results of the management cannot be obtained if this modification is continuously followed. Although accurate determinations of the blood carbonates and all other evidence indicate that the continued use of alkalies in no greater quantity than that required to control the free hydrochloric acid is not harmful, it has been our custom to stop all powders for five days at the end of ten weeks and then resume them as before for five or six weeks. Then stop them for five days, then resume them for five or six weeks, thus accurately controlling the acidity continuously with the exception of the five-day period every five or six weeks until the treatment is to be discontinued. During the five days when no powders are taken the frequent feedings should be continued and without change. If during this five-day period the bowels do not move on a given day a small enema of warm water may be used the following morning.

"During the three or four weeks that the patient is under the careful supervision of the physician the quantity of alkali required to control the free acidity will have been determined; the patient learns how to manage his diet and powders so as to adequately control the free acidity. Above all he learns why he is faithfully to continue to do his part. Experience with large numbers of all classes of patients has abundantly demonstrated that it is easily possible to continue with the hourly feedings and with the powders while the patient is attending his regular work. Thus the ideal conditions for healing may be maintained for as long a period as desired. An ulcer that upon roentgen ray examination shows no evidence of large size or other unfavorable conditions is likely to heal relatively rapidly under the influence of the management advocated. Because of the seriousness of the disease, however, and the relative ease with which the favorable conditions for healing may be maintained, the accurate management should be continued as outlined for as least one year. No possible harm can come from continuing the management for months after the ulcer has healed. The cost in time and trouble to the patient is insignificant when compared with the permanent relief from the distress and dangers that attend an unhealed ulcer."

In chronic ulcer which has assumed the pyloric obstructive type Sippy's experience prompts him to state that many such cases will yield to faithful persistent dietary and alkaline treatment and that in a goodly number gastro-enterostomy is finally found to be unnecessary. It is extremely difficult to control all patients absolutely over a protracted period of treatment and those who will not or cannot afford or submit to such control would better have gastro-enterostomies or possibly pyloroplasties. It should be remembered, however, that some patients after gastro-enterostomy experience a return of ulcer symptoms, even including high acidity and bleeding. Therefore, surgical measures, though a boon to the majority of chronic obstructive ulcer cases may not be depended upon to be an absolute cure in every patient. Many physicians have been called to treat ulcer cases at a more or less remote period following gastro-enterostomy. Some of these prove to be jejunal or gastro-jejunal ulcers but many are recurrences of old gastric or duodenal ulcers.

Medical Treatment.—For many years **bismuth subnitrate** has been recognized as one of the most valuable, if not the most valuable, medicinal remedy in the treatment of ulcer, and Chas. D. Aaron particularly emphasized its importance. In the acute and non-indurated ulcers it is a most efficient drug but should be given in very much larger doses than those ordinarily employed. A pure bismuth subnitrate or subcarbonate may be safely given in teaspoonful doses four or six times a day for several days, and half teaspoonful doses may be employed for weeks or months without harm in the great majority of cases. It is a good plan to give a level teaspoonful stirred in one-third of a glass of water half to one hour before breakfast, and during the digestive period of the day to give **cerium oxalate** and **calcined**, or the carbonate of **magnesia** with it. This combination is utilized in Stockton's "gastric sedative" which consists of cerium oxalate, one part, bismuth subcarbonate, two parts, and calcined magnesia or the light carbonate of magnesia, four parts. The dose of this is ordinarily one teaspoonful stirred in one-quarter or one-third glass of water as often between feedings as necessary to neutralize acidity and keep the ulcer area more or less coated with the soothing protective and healing bismuth. The magnesia often proves too laxative and it is best to add calcium carbonate to the mixture, usually the same quantity as the bismuth content; or bicarbonate of soda may be added. Sometimes but little magnesia is tolerated by the patient owing to its laxative effect and then more calcium carbonate and bismuth may be substituted. It is unfortunate when the magnesia is not tolerated if the gastric acidity runs high as it binds more acid than any other alkali employed. Bicarbonate of soda affords quick relief in the hyperchlorhydria of ulcer but its acid binding property is less than that of calcium carbonate and magnesia and it seems to provoke the secretion of more acid. It is taken too frequently and too copiously by many ulcer patients and in the histories they relate there will generally be found reference to a very frequent resort to the use of soda with an account of prompt but temporary relief. If it is employed it is best to restrict its use to small doses and to combine it with bismuth and magnesia. An alkaline mineral water, especially Vichy and Contrexville affords an excellent method of using it in weak dilution. **Kaolin** is a remedy which has somewhat recently been employed internally in the treatment of ulcer. It may be combined with soda, bismuth and magnesia or it may be given in two or three gramme doses stirred in milk, water or a demulcent such as flax-seed tea or slippery elm water.

In order to lessen gastric secretion and overcome pylorospasm **belladonna** or **atropin** have been much employed and in some cases distinct relief attends their use. They are, however, often disappointing. If the acute stage of ulcer has passed and there is no further evidence of bleeding, yet gastric hyperesthesia is troublesome, **bromids** may be given with belladonna, as Friedenwald advocates, in the treatment of non-ulcerative hyperchlorhydria and pylorospasm. Macht has contributed to our knowledge of the action of papaverine upon pylorospasm and his synthetic preparations benzyl-benzoate are used in some cases. Local anesthetics, **cocaine, chloretone** and **anesthesin**, are sometimes employed for the relief of pain and spasm. They occasionally afford comfort and may be combined with bismuth or other soothing medicaments. The late John Musser called attention to the use of hydrogen peroxide in hyperchlorhydria many years ago before it was realized that ulcer so commonly accompanied this secretory state. He found that if it was given undiluted in drachm doses gastric acidity was notably reduced. The depressing effect of **fats** upon acid secretion was noted some years ago and **ceram** is very useful in the management of ulcer. As a soothing protective medicament in ulcer purified vaseline or liquid petrolatum seem grateful in some cases. If slight hemorrhage persists **suprarenal extract** in .06 gram (.9258 grains) doses may be given thoroughly rubbed up with bismuth in konseals or capsules; or **silver nitrate** may be employed in doses of .015 gram (.23145 grain) every four hours for a few days.

Resorcin in .06 gram (.9258 grain) doses and ichthyol .20 to .30 grams (.086 to 4.62 grain) doses in capsule may be given in the hope that they may stimulate epithelial regeneration and healing.

These drugs are mentioned as being safe and proper to use in ulcer cases but less reliance is placed upon them as our experience with ulcer widens. It is now recognized that most ulcers which will not heal under duodenal feeding, rest, diet and alkalies with bismuth, eventually require surgical intervention, but in all fairness medical treatment should be thorough, systematic and prolonged; the patient's sincere and faithful co-operation is also essential.

In the treatment of peptic ulcer with pyloric obstruction, Sippy carries out in principle the same medical plan as that adopted for the treatment of the non-obstructive type and he says that the management differs only in the following particulars: In many cases greater quantities of alkalies are required and the stomach is washed out every night half an hour after the last alkaline powder is given. If an excessive night secretion is present the stomach is again washed out at midnight and Sippy says that if 100 c.c. or more of strong gastric juice is present, it results from continued secretion; therefore, after the aspiration of the gastric contents at 9:30 P. M., an alkaline powder should be given and repeated every 30 or 40 minutes thereafter until midnight at which time the stomach is again aspirated. Enough alkali should be given to control the acid until the midnight lavage. Sippy says that "the night secretion usually rapidly disappears when the free acid is thus accurately controlled," and that within a week not more than 15 to 20 c.c. of secretion is found at midnight or thereafter. This small quantity need not be removed or controlled by alkalies. Subsequently the stomach should be emptied by means of the tube one-half hour after the last powder is given. He has found that if it is possible to control the acid as above outlined at night, it is likely to be controlled during the day, but an afternoon aspiration may be done before an alkaline powder or food is taken and the acidity tested. In chronic obstructon of marked degree, the powders after the 9:30 P. M. aspiration should be continued and at 11:30 P. M., another aspiration should be given for two or three weeks at which time excessive night secretion may be controlled. In some cases he gives an alkaline powder every hour during the night for awhile and considers that this removes the tendency to excessive night secretion. In about two weeks from the beginning of the treatment a seven-hour motor meal may be given to determine the emptying power of the stomach. He does not give alkalies during the seven-hour period after the motor meal. The intake of food and liquids should be compared with that which is removed by lavage. The aspirations serve three important purposes: "First, the aspiration one-half hour after the last powder is taken removes all food and secretion and thus lessens the stimulus to a night secretion with its attending irritation and possible corrosion. Second, aspirations and tests for free acid enable one to maintain an efficient neutralization of the corrosive action of the gastric juice with a minimum use of alkali. Third, the absence of occult blood upon repeated examination of the aspirated stomach contents enables one to practically exclude a cancer of the stomach developing at the seat of a peptic ulcer. The continued absence of occult blood in the stomach contents combined with its presence in the stool for a time after which it gradually disappears under treatment renders duodenal location of the ulcer probable.'' Sippy classifies the clinical types of pyloric obstruction as: First, that which disappears during the first three weeks under the influence of his medical management and he says that ''between 80 and 90 per cent. of all cases of pyloric obstruction due to ulcer belong to this type.'' Obstruction is caused by either pyloric spasm, acute inflammatory swelling and, in rare instances, a local peritonitis, therefore, it is unwise to conclude that cicatricial stenosis exists when, upon a first examination, stagnation of gastric contents is found as ''a pyloric obstruction of the highest grade as evidenced by pronounced undulations of the abdominal wall due to vigorous peristaltic contractions of the stomach, the presence of sarcinæ in abundance and the retention of food from 12 to 36 or more hours, may yield so rapidly that after one or two weeks' management the stomach is found empty 6 to 7 hours after the ingestion of a motor meal. Under such circumstances a tissue narrowing of serious grade may be excluded.'' Second, in the classification, come those cases in which the evidences of pyloric obstruction do not disappear during the first three weeks of the medical management outlined. In these cases seven hours after the motor meal, 100 to 200 c.c. of food and secretion are still present. ''Approximately, 15 per cent. of all cases of pyloric obstruction due to ulcer belong to this type'' and, other things being favorable, a gastro-enterostomy by a ''fully qualified surgeon,'' is indicated. In some of these cases, however, if the medical treatment is carried over a period of two or three months, a motor meal will be completely evacuated in seven hours. Sippy says this occurs ''in more than one-half of this type of cases.'' The improvement largely results from reduced swelling of the pyloric mucosa and a reduction in the inflammatory state of the tissues. It is maintained that carcinoma of the stomach is more accurately determined by this watchful and systematic management of the obstructive type of case. If there are no complications such as carcinoma of the stomach or gall-bladder or appendix disease, the relief afforded by the medical management is definite and significant. Possibly abdominal distress may occur because of irritation of the colon by too much magnesia. In those cases of the obstructive type of ulcer in which the medical management is successful and in which the motor function of the stomach is restored apparently to normal; in which there is no evidence of further bleeding; in which there is continued comfort, the diet is to be observed and the alkaline powders are to be taken the same as in cases of the non-obstructive type, but the patient should be taught to wash out his stomach every night until such time as, on the simple diet, he finds it approximately empty at 9:30 or 10 o'clock. The evening meal should always be light. ''The patient is instructed to test the ability of the stomach to empty itself about once each month using an ordinary full meal.'' Sippy deprecates untimely gastro-enterostomy and his splendid writings show that the operation should not be performed until his thorough medical management has been carried out a long enough time to make sure that the gastro-enterostomy absolutely is necessary.

Further Considerations as to Dietary and Medicinal Treatment.—Rest in bed for several weeks is particularly indicated for those cases showing decided loss of weight with a persistence of disabling gastric distress and for those with pronounced pain or hemorrhage. For others ambulatory treatment suffices if rest is taken for an hour after each meal and the diet is fastidiously observed and the medicines accurately taken. There is no rule which applies unmodified to all cases; individual study is required. Some patients progress most comfortably and favorably upon a semi-solid meatless diet while others do much better on chicken, fish, ham, lamb and beef. Friedrich Muller advises against the use of fat meats, thick soups, gravies, goosemeat and brain tissues but allows cream copiously for its high caloric value and pain controlling property. For pain, he says, resort may be had to silver nitrate 0.2 gram in 150 c.c. of distilled water in doses of one tablespoonful before meals for three weeks with bismuth subnitrate or subcarbonate in teaspoonfuls stirred in water before meals. He prefers the atropin derivative eumydrin to atropin and advises its use in doses of .002 gram twice daily either in pills or solution. He advises giving papaverine .03 or .04 gram twice daily.

The use of astringents, alkalies and belladonna is advocated by L. von Friedrich in the treatment of gastric hyperacidity. A combination called belladonna-neutralon is employed in tablets containing 1.5 gram neutralon and extract of belladonna .009 gram. Astringents cause the absorption of pepsin and with the action of the alkaline content and belladonna a reduction in acidity and pepsin values is accomplished.

In the older medical literature dietetic treatment was taught but the physicians in those days lacked the modern knowledge of the refractory nature of ulcer and of the extreme difficulty of determining whether, or when, an ulcer had healed. Dietary restrictions were not enforced over a long period nor were they rigid enough to insure against recurrence. Present day writers at home and abroad recognize the extreme importance of rigid dietary treatment over a long period.

J. Schryver considers a milk diet as ideal, but the caloric needs of the patient should be estimated not only to avoid undernourishment but because food requirements which are unsatisfied cause increased gastric unrest.

For the first week milk alone is given, as at that time pain and bleeding will usually have disappeared, then milk and thin milk porridge, groats, maizena, ground rice, and rolled oats all boiled in milk are allowed. At 8, 12 and 6 o'clock a soup plate of porridge with butter and sugar is given and after two hours a glass of milk is given every hour until the next porridge is due excepting after 10 P. M. At the end of a week the feces is examined for blood and if it is found present the patient is kept in bed two weeks longer. During the third and fourth weeks milk, buttermilk, sour cream, sweet cream, water chocolate, milk chocolate, weak tea, water (with the addition of a little bicarbonate of soda), biscuit, toast, much butter, sugar, molasses, honey, jam without whole fruit, soft-boiled eggs, thin porridge, milk soups, peas, beans, cauliflower tops, carrots, spinach, stewed fruits (all mashed), finely chopped beef, boiled fish, dry biscuit, sweet chocolate. After 4 weeks the patient is allowed out of bed but must conform to the dietary rules for a year. If a relapse occurs after that Schryver says surgical measures are called for but that is not necessarily so as relapse may be successfully treated along medical lines.

It will be seen that dietic measures together with the persistent use of alkalies and bismuth are here given emphasis and repetition because it is felt that much more may be accomplished by medical treatment than the surgeons and many physicians realize. This, of course, applies to acute ulcer and to ulcers which, though chronic, have not developed a medically incurable condition. The great trouble with most ulcers medically treated is that they are not healed when either the patient or the physician or both abandon treatment, and they are therefore bound to recur or at least to occasion an exacerbation of symptoms again, which is termed a recurrence. For that reason an ulcer treatment must be checked up cautiously as to cessation of occult bleeding. Radiographic illustrations of ulcer cures are now quite commonly shown, but though radiography demonstrate a scar after medical management it does not follow that it represents an unhealed ulcer.

A very good plan of treatment frequently adopted by the author is the following: If the case is a suitable one and the duodenal tube is taken and tolerated comfortably it is employed for feeding. Two hundred and fifty cubic centimeters of certified milk is given by the drop method every two hours from 7 A. M. until 9 P. M. This is continued for two weeks. Milk of magnesia is given as a laxative and antacid. Bismuth and chalk and cerium oxalate have been found to clog up the perforations in the bulb. After duodenal feeding is discontinued, milk, eggs, cream of wheat, huskless oatmeal, farina, thoroughly cooked boiled rice and milk toast are allowed. Baked and boiled custards, light and flaky freshly made cottage cheese with thin white bread and butter are permissible. Bread pudding, rice pudding, tapioca and sago puddings and ice cream and sponge cake are included. At first small amounts are served by the nurse but these are daily increased until a sufficiently high caloric value is reached. At the expiration of two weeks, that is four weeks from the beginning of the treatment, vegetable purees, white, mealy, thoroughly cooked, baked potatoes and finely minced or ground tender chicken and lean meats are allowed. Later tender fresh fish and fruit jellies are added to this diet which is ordered continued for a year if nothing arises which may require its alteration.

From the beginning of treatment, if no recent hemorrhage has occurred, light poultices made of one-third flaxseed meal and two-third bran are placed over the stomach every two, three or four hours according to the amount of pain and pylorospasm there is present. The skin is prepared for the poulticing as directed by von Leube. The poulticing is continued during the day for two weeks and then the frequency is lessened and when the patient is allowed out of bed it is discontinued unless necessary at times for pyloric spasm and pain.

When duodenal feeding is not employed the patient is given rest in bed and if there has been recent hemorrhage no food is allowed by the mouth for two or three days, then milk diet is given for three days from two to three pints being given each day. Thereafter for a week the milk is given and beaten eggs are added, at first one, then two, three and four a day up to the end of the second week and then the diet as outlined for use after the cessation of duodenal feeding is allowed.

From the outset in those not receiving duodenal feeding as large quantities of magnesia, soda, calcium carbonate, bismuth, cerium oxalate and kaolin as are necessary to neutralize gastric acidity and coat, protect and sooth the ulcer area are given. The gastric contents are examined at times to ascertain the efficacy of the alkaline medication. One teaspoonful of bismuth subnitrate is given each morning when the patient awakens and one hour after the first food a teaspoonful of the following prescription is given stirred in a quarter of a glass of water:

Cerii oxalatis, 1 part.
Bismuth subcarbonatis, 2 parts.
Magnesia carbonatis levis, 4 parts.

An hour later another teaspoonful of this powder is given and in another hour fifteen grains of calcium carbonate and the same amount of soda bicarbonate stirred in a quarter of a glass of water. In the afternoon this procedure is repeated and again in the evening. At bedtime another teaspoonful of bismuth subnitrate is given and if there is pain and discomfort in the night the gastric sedative above is given, sometimes with, sometimes without, the addition of half a teaspoonful of kaolin. It is necessary to withhold the magnesia and give more calcium carbonate if the gastric sedative proves too laxative. Sometimes the calcined magnesia is used in place of the light carbonate; it has more acid binding power but is not quite as pleasant to take. By the very liberal use of alkalies the gastric acidity is greatly reduced, pain is overcome and healing of the ulcer promoted. With the exception of soda bicarbonate the other drugs are insoluble and are for the most part not absorbed; acting only locally in the gastro-intestinal tract. The great majority of patients with juxtapyloric ulcer, either gastric or duodenal, are quite relieved by this treatment and gastric analyses, thread tests and radiographic studies confirm the clinical concept that the ulcers have healed. The hurry and distraction of a busy internist is apt to make him forget that in an ulcer case a subsidence or disappearance of symptoms is not sufficient warrant for him to relax his instructions or control which should always be based upon the assumption that there is an ulcer area, probably healed, possibly not, which is there whether he sees it or not, whether the patient feels it or not, and his advice must be given accordingly. It is the only safe way from the medical standpoint.

There is, however, another viewpoint from which to consider the question of prolonged medical versus short surgical treatment and this is well put before us by Wm. J. Mayo who says that more than 90 per cent. of cases coming to them are wage earners unable to afford the financial demands of prolonged medical care. Furthermore their social and living conditions militate against proper dietetic regimen. Wage earners with few exceptions are forced to continue work and a large percentage of those seeking medical or surgical advice have already suffered disability, loss of weight and strength, and loss of income owing to the illness from which relief is sought.

The home conditions, habits, environment and occupation, as well as the financial ability of a patient should be fully investigated before the protracted course necessary for a medical cure is advised for him. Mayo asserts that their surgical treatment is satisfactory in 95 per cent. of cases of duodenal ulcer and 85 per cent. of gastric ulcer.

The author, however, is convinced that ulcers, even though occurring in wage earners, which after proper study are evidently suitable for medical treatment should be given that form of treatment, preferably in a hospital, before being subjected to surgical treatment, as it will be found in a large number that a short course of treatment will give comfortable relief and that work may be resumed if the patient will follow his dietary and medicinal instructions; furthermore no recurrence will occur in a considerable proportion of the cases. This opinion is also held by Dr. Franklin W. White.

CHRONIC ULCER.—At the onset it is most important to determine whether or not a chronic ulcer is amenable to medical treatment, whether it may be properly and conscientioously treated medically. Many may differ with Sippy in his care of the obstructive type of ulcer, but his experience has been duplicated in cases under our observation and care. It is, however, rarely possible to undo cicatrical pyloric stenosis by medical management. To be sure we see a return of the propulsive power of the stomach under diet and lavage and antiphlogistic, antacid, astringent and antispasmodic remedies, but in a few days, or any day, stagnation is apt to manifest itself again. Cases with cicatricial pyloric stenosis need either a modified pyloroplasty or a gastroenterostomy.

If a chronic ulcer is situated far enough away from the pylorus not to produce stenosis at that point, it should be determined if possible how deep, large and indurated it is, and especially whether it is of the perforating type, or whether it has perforated slowly and fastened the stomach to a neighboring tissue.

Uncontrollable pain usually indicates the existence of a penetrating ulcer if syphilis be excluded, and after thorough medical treatment fails to relieve such pain operation will usually disclose an ulcer presenting characteristics precluding the possibility of its cure by medicines, rest and diet. A chronic ulcer, the symptoms of which yield to treatment with reasonable promptitude, is in all probability, not of the incurable type and the dietary and medicinal management above outlined carried on for many months usually suffice.

Pathological Changes.—In reference to the location of gastric ulcer Sippy states that in over three thousand autopsies the following was found:

Lesser curvature, 35 per cent.
Posterior wall, 30 per cent.
Pylorus, 12 per cent.
Anterior wall, 9 per cent.
Cardia, 6.5 per cent.
Fundus, 3 per cent.
Greater curvature, 3.5 per cent.
Anterior and posterior wall, 1 per cent.

Acute or Recent Peptic Ulcer.—Acute peptic ulcer, like the chronic form, is found most commonly on the posterior surface of the stomach near the lesser curvature. It may be nearer the pylorus or, indeed, it may occupy a position at the pyloric ring although in the terminal three-fourths of an inch of the prepyloric part, the Canal of Jonnesco, ulcer is rare. In the duodenum it is usually situated in the first portion though it may be found two inches or more beyond the pylorus. In the stomach it is sometimes present high up toward the cardia on the posterior wall near the lesser curvature or it may occupy a position in the anterior wall of the stomach either near by or farther away from the pylorus. Fundus ulcer on the anterior wall is rare. Ulcer near the cardia is comparatively rare. Acute ulcer is superficial and does not extend deeply. It is round or oval, occasionally somewhat irregular in shape. It is nearly always single though two ulcers are occasionally found and more than two in rare instances. In the acute form the muscular coat is seldom penetrated. The size of the ulcer varies from that of a few millimeters to one or two centimeters in diameter; occasionally ulcers are as large as a silver half dollar. The edge of the ulcer is clean cut and reddish in color, there is but little swelling or infiltration of the tissues of the border and the ulcer surface is free from granulations. At the edges of the ulcer and extending a few millimeters beyond there may be ecchymosis but that is rather the exception. The factors which initiate acute peptic ulcer are a mystery to us, speculate as we may. The author is old-fashioned enough to liken the irritable superficial gastric ulcer to many of the clean-cut sores seen in ordinary cancrum oris, the great difference being however, that the gastric lesion is usually single and the cankered mouth usually presents many sores. The clean edged sore in the mouth strikingly resembles the classic description of the superficial gastric ulcer. Furthermore, as the gastric lesion may pass into the chronic stage so the buccal sore often becomes deep, painful and obstinate and sometimes leaves a scar.

It is not possible to tell exactly when an acute ulcer develops. It may or may not give rise to immediate symptoms. Possibly many small ulcers form and heal with only a short period of irritative gastric symptoms, possibly none whatever. Hemorrhage may be the first symptom. Whatever the process, whether it be sudden tissue autolysis following a herpetiform lesion or whether the changes are those of a slower ulcerative process, the blood-vessels are eroded and hemorrhage may occur. It is thought that the cases of sudden unheralded hematemesis are not infrequently due to the hemorrhage of gastric erosion described by Dieulafoy and which he distinguished from Cruveilhier ulcer. At all events, such a single gastric hemorrhage occurs in some cases and the patients make rapid recoveries without subsequent symptoms of ulcer. An ulcer which does not rapidly heal soon assumes more or less induration of its borders and tends to involve deeper tissues, penetrating the muscularis. Although the pathologic changes are not those of a typical inflammatory process there is more or less round cell infiltration and fibrous increase as time goes on. The ulcer may present a terraced appearance or the edges have a "pouched out" appearance from the mucous surface to the bottom of the ulcer. When the terraced or step-like sides are seen the shape is more or less conical or funnel-shaped. The whole ulcer surface is usually clean. If the deeper layers of the gastric wall are reached by the process there is a peritoneal reaction and a fibrinous layer is formed over the portion underlying the ulcer on adhesions may be found in the resulting local perigastritis. These are changes, however, which are a part of the process of chronic ulcer.

CHRONIC ULCER.—Chronic ulcer is not a continuous pathologic process in all cases. It may permanently heal with more or less scar tissue production, or it may heal temporarily only to break out again and make additional invasion in one or another phase or direction leaving a greater proliferation of scar tissue or involving a greater area in an irregular form or tending to perforation and thereby causing more local peritonitis and adhesions to the liver or gall-bladder or biliary ducts or the intestines, inducing symptoms referable to such involvement. There are changes suggesting infection and inflammatory processes. The ulcer may grow very deep and its margins markedly indurated, the hardening and thickening extending considerably beyond the ulcer margins. The blood-vessels undergo thrombosis and some are obliterated while others are open, eroded and unable to contract so that hemorrhage may be severe and prolonged. Healing granulations are prevented by the continued action of the acid gastric secretion and healing is prevented as proliferation of embryonic elements results only in further thickening and damage. Stockton well describes the changes incidental to chronic ulcer in the following words: "The mucous glands and the specific glands of the stomach, as well as the muscle fibres, are atrophied and their arrangement disturbed by the crowded-in of round cells and by the fibrous transformation that follows. In the midst of this there are seen areas of recent lymphangitis. The walls of the blood-vessels are atrophied and infiltrated by embryonic cells, definite endarteritis is present. This is sometimes obliterative in character, at other times small aneurisms develop. Insofar as the arterial changes lead to obliteration and thrombosis, there is a decrease in the tendency towards hemorrhage. On the other hand the tissues thus subjected to a diminished blood supply are more vulnerable to the digestive action of the gastric juice."

An appearance of carcinomatous metamorphosis may result from epithelial proliferation with fibrous induration and in such areas of chronic gastric ulcer actual malignant changes sometimes develops. The Mayos have particularly made this clear to the profession. In 54 per cent. of 69 cases they operated upon in 1905 and 1906 cancer was found to have its origin in ulcer. Such a frequency of carcinomatous metamorphosis has not been found by other observers. In "An analysis of one hundred and eighty cases of Cancer of the Stomach" S. Paul Taylor and T. Grier Miller concluded that the incidence of carcinoma upon an ulcer basis did not exceed 17 per cent.

Figures show considerable variation and some clinicians regard the development of cancer upon ulcer as rare and maintain that "once an ulcer always ulcer" is the rule. Osler called attention to the danger of mistaken pathologic diagnosis because of the close resemblance sections from some ulcer areas may bear to carcinoma. Expert pathologists are not apt to misinterpret the pathologic sections and as such experts have often found distinct evidences of malignancy in old ulcers we must admit that cancer has its origin at the seat of chronic ulcer in a considerable proportion of cases.

Multiple ulcers may result in far spread damage and deformity. An ulcer may be present in the stomach and another in the duodenum. Strange contractures of the stomach may develop and irregular pouching and band formation may be found. From a proximal pouch gastric contents resembling those extracted from a dilated esophagus may be withdrawn while from a distal portion highly acid contents may be derived, and in case of an associated pyloric stenosis characteristic contents indicate of food stagnation may be recovered. In one of the author's cases extensive multiple chronic ulcers had greatly reduced the capacity of the prepyloric and middle portions of the stomach in so far that only a narrow stream of barium was seen from the fundus to the duodenum. The resemblance to linitis plastica was very marked and the woman had been able to take only small quantities of liquid foods for many months, her history of gastric disorder including hemorrhage dating back many years.

DUODENAL ULCER

Although gastric and duodenal ulcers have much in common and are considered together by some writers, there are many distinctive features pertaining to duodenal ulcer which warrant its separate consideration. It is more frequently met with in men than in women; it is more constantly associated with a high gastric acidity than is ulcer of the stomach unless that be located near the pylorus; it is accompanied more steadfastly by typical hunger-pain than is the usual gastric ulcer; it is much less frequently attended by hematemesis than is gastric ulcer. Among its symptoms vomiting is noted less frequently than is the case with gastric ulcer; it does not so often occasion the gastric nor the perigastric deformities which so frequently result from gastric ulcer; it is rarely the seat of malignant metamorphosis and on the whole may be said to run a milder, less disabling course in many cases than gastric ulcer. There are exceptions to some of these characteristics. For instance, copious hematemesis may, in exceptional cases, attend duodenal ulcer owing to the regurgitation of blood into the stomach from flooding of the duodenum by a copious hemorrhage, but in such cases there is apt to be a diarrhœa with profuse bloody stools. If the patient is nervous and gastric hyperesthesia is marked vomiting may occur quite frequently. In some instances instead of running a course mild enough to allow the patient to continue his occupation the affection assumes most troublesome and disabling aspects.

Etiology.—So far as we know the etiology does not differ materially from that of gastric ulcer though its greater frequency in males may have some bearing and it, at all events, leads to considerable etiologic speculation.

The anatomical difference between the position of the duodenum in the male and that of the female supposedly renders the former the more susceptible to ulcer. The duodenum in the males ascends sharply in its first portion and its second descends to the common bile duct, whereas in the female the first portion lies more nearly transverse. It is argued that because of this arrangement the bile and pancreatic secretion bathes the first portion of the female duodenum with an alkalinizing and neutralizing effect whereas in the male the first portion is more constantly acid and subjected to the corrosive influence of the gastric secretion. If the male duodenum is thus handicapped it would seem to emphasize the importance of rest in bed as a part of treatment, thereby favoring the contact of the neutralizing secretions with the juxtapyloric part.

A peculiar feature in relation to the etiology of duodenal ulcer is its occurrences after extensive cutaneous burns. It has been proposed that some toxic substance is secreted with the bile after burns and this injures the duodenal mucosa, or that antiferments present in the mucous cells are destroyed by severe burns of the surface of the body. Toluy lendiamin injected hypodermatically in dogs has been followed by duodenal ulceration. The true explanation of the association of cutaneous ulceration has not yet been made but septic embolism or thrombosis of the vessels of the mucous membrane seems the most reasonable.

The ulcerations differ from peptic ulcer. They occur in 6.2 per cent. of fatal burn cases. They are found somewhat more frequently in young females. They are often multiple, two to six, sometimes accompanied by gastric ulcer, and develop about ten days after the burn, rather more frequently after burns of the trunk than the extremities. They are often found in the lower portion of the duodenum and vary in width from 1 to 5 m.m. and in length 5 to 15 m.m.

Duodenal ulcer is occasionally found in infancy. Peterson reports two cases discovered in a series of 566 autopsies. Digestive disturbances and lowered general vitality with tarry stools, if intussusception and colitis are excluded, should arouse suspicion of ulcer.

In reference to the etiology of duodenal ulcer the experimental observations of Frank C. Mann and C. S. Williamson of the Mayo Clinic are well worthy of particular attention. They have succeeded in causing 90 per cent. of ulcers in the jejunum following the drainage of the duodenum into the ileum and the anastomosis of the jejunum to the pylorus. The operation transfers the alkaline flood from the bile and pancreatic ducts to the ileum, and the jejunum attached to the stomach is subjected to the full erosive force of the acid output from the stomach. The jejunal ulcers have formed within three weeks of the operation, some were single and some were multiple. The pathologic changes were distinctly those of peptic ulcer. The work emphasizes the relationship of high gastric acidity, which results in excessive doudenal acidity, to the incidence of duodenal ulcer although Dr. Mann is conservative and reserved in his opinion as to this etiologic relationship.

Diagnosis.—It is important to differentiate clinically between duodenal and gastric ulcer, and the most helpful diagnostic points are these: The duodenal ulcer is more frequent in the male sex, the characteristic hunger-pain (not the pseudo-type) and the location of pain and tenderness to the right of the median epigastric line, the almost constant superacidity of the gastric contents and the frequent absence of blood or occult blood therein, while the presence of blood or occult blood is noted in the duodenal contents and in the feces, the positive bloodstain on the bile-colored portion of the Einhorn thread are important. Added to these we may have the radiographic evidences of gastric hypermotility, pylorospasm, deformity of the cap and a six-hour gastric residue, with, in some cases, presumptive evidence of adhesions and fixation.

We should remember, however, that gastric and duodenal ulcer have been found synchronously in about six per cent. of all cases seen at the Mayo clinic. In the usual event of encountering one of these cases most careful studies will be called for in order that a correct diagnosis may be accomplished; as it is probable that it has been made most frequently at operation.

Recently G. Singer has emphasized the role played by vague and circulatory influences in the pathogenesis of duodenal ulcer which is considered a secondary disease occurring as a reflex along the path of the vagus. Attention is directed to the incidence of duodenal ulcer in connection with meningeal or brain disease, tuberculosis or other diseases of the lungs and bronchi, gall-bladder disease and cirrhosis of the liver. Tuberculosis ulcer of the duodenum may occur. Lead poisoning may cause a neurogenous duodenal ulcer through a vagus neuritis. Heart disease has long been noted in association with ulcer.

It is very probable that several factors are synchronously influential in the causation of ulcer and this has been recognized by clinicians for many years. It is well that we are reminded of the fact occasionally as there has been a tendency manifested of late to attribute all ulcers to the result of some focal infection and true and urgent as that contention is yet there are other agencies which are often operative at the same time.

In a valuable contribution recently Bevan cited the enormous increase in peptic ulcer in Central Europe during the last war. Anemia, worry and nervous strain are potent factors in disturbing the digestive functions, exciting hyperchlorhydria and pylorospasm and playing a detrimental rôle toward trophic control and circulation. The remarkable tolerance of the digestive apparatus toward coarse and indigestible foods is a factor of safety in health but is broken down by various physical and nervous conditions such as frequently antedate the development of ulcer.

Throughout the many past years during which ulcer has been studied and discussed there has always issued forth the statement that its etiology is unknown and therefore prevention is hindered and treatment unsatisfactory. A pessimistic note has, for the most part, pervaded the recent papers and discussions on ulcer. Why should discouragement thus prevail amongst us with regard to our knowledge and treatment of ulcer? Great good and many cures are, and have been, effected by medical and surgical treatment of ulcer. Long ago von Leube reported upwards of 90 per cent. cures and from Moynihan numerous cures come, and from the Mayos upward of 90 per cent. cures, and from Sippy the statement that the great majority of ulcer cases are cured by his systematic plan of medical treatment, and from many other gastroenterologists reports of their satisfactory results in the treatment of ulcer. The writer's experience, covering many years, has taught that ulcers and their complications have been cured in a very large proportion of all cases studied and treated by medical and surgical procedures. A disparaging and discouraging view of the whole matter is unwarranted.

The radiographic observation of duodenal ulcer is as follows*:

> The radiographic examination of the duodenum for duodenal ulcer is perhaps best accomplished by the plate method for the reason that it is quite difficult to visualize the duodenum for any great length of time so that its contour can be carefully studied. It should of course, be studied by fluoroscopic means which gives us its location and occasionally one can clearly demonstrate a persistent deformity.
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> However, in the serial plate method taken in different positions the most important sign is the deformed duodenal cap, with as a rule excessive motility of the stomach and a rapid empyting of the stomach's opaque contents. The deformity usually persists in all stages of the examination As most of the duodenal ulcers occur in the first portion of the duodenum, the deformity can usually be demonstrated on a plate if the technique has been correct.
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> When the first portion of the duodenum is normal, and is shown to have the characteristic shape and regular outline we can rule out ulcer of this portion, while a constant defect of the cap means pathology of this portion of the duodenum; however, this deformity may mean ulcer, adhesions or pressure from neighboring organs anyone of which may produce this deformity. Adhesions frequently form from a pathological gall-bladder.
>
> An indurated ulcer of the duodenum will cause deformity of the cap, while an ulcer, which is a simple erosion of the mucous membrane will not show a deformity.

Treatment.—The treatment of duodenal ulcer differs in no essential respects from that of gastric ulcer. Dietetic restrictions are the same, the need for alkalies and protective, slightly astringent remedies is the same foci of infection should, if possible, be abolished, the teeth and mouth require scrupulous care as in gastric ulcer. Roentgen ray therapy has been employed in the treatment of duodenal ulcer, 25 to 35 Milliamperes, 80 Kilovolts, 3 mm. Aluminum filter, skin target distance 10 inches. Exposure at intervals of from four days to two weeks were made over the stomach. After four treatments the cases were clinically well. When one recalls the decided effects of the x-rays upon glandular structures, the thyroid, ovaries, testicles and lymphatics, it would seem advisable to proceed very cautiously as damage to the pancreas may result. Furthermore it is probable that clinical cure occurs in some cases of duodenal ulcer with little or no treatment of any sort, and there seems to be no sound reason why this treatment should be employed when the excellent results of the above-mentioned plans of therapy are recalled.

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*This radiographic discussion was kindly supplied by Dr. Edward Charles Koenig, referred to previously.

Pathology.—The pathologic changes brought about by chronic duodenal ulcer are different from those of gastric ulcer. Most duodenal ulcers have no craters and are not of the callous variety but appear more like erosions. Some, however, on the posterior wall have craters and are indurated and periduodenitis entails pathologic changes mentioned elsewhere.

If perforation occurs it may be impossible to determine from the symptoms produced whether the catastrophe is due to a gastric or duodenal lesion unless opportunity for a differential diagnosis is had prior to its happening. In the common run of cases the chronic form may result in some degree of induration, scarring and deformity, of the duodenum, and in the formation of adhesions to the gall-bladder, gall ducts, pancreas, liver, colon, omentum, mesentery or the retroperitoneum.

GASTRIC AND DUODENAL EROSIONS

Etiology.—During the process of gastric lavage occasionally small fragments of mucous membrane are found in the washings. They are usually soft and of a pale pinkish appearance. They are sometimes found in cases with achylia gastrica and in such Einhorn noted the presence of apparently normal glandular structure thereby being enabled to state that all cases of suspension of the gastric secretory function were not associated with complete parenchymatous atrophy. On the other hand some fragments of mucosa washed up in cases of serious impairment of the gastric secretory function were found to present the pathologic appearance of glandular atrophy. It was apparent that the tissue resistance of the gastric mucous membrane was distinctly lowered in some cases studied. There appeared to be a softening (gastromalacia) of the mucosal structure in some instances, while in others the usual pathologic picture of gastritis, with glandular atrophy, altered epithelium, and fibrous hyperplasia was present. The outstanding feature from the practical viewpoint is that there exists a markedly increased vulnerability of the mucosa in this complex and it is much more easily traumatized than in a healthy state. Fragments have been found in the washwater in cases of high acidity. The question of careless and unnecessary traumatism must here be mentioned. It is probable that many fragments of the gastric mucosa have been torn off by rough usage and suction. If the stomach tube is pinched and quickly and forcibly withdrawn a bit of the mucosa is very apt to be torn away by suction. The bleeding following this rough treatment is fortunately insignificant as a rule and the wound heals promptly. In a few cases a sense of gastric soreness may be complained of for a few days. It is remarkable that these wounds seldom develop into peptic ulcers.

Aside from the above there are well-authenticated illustrations of smaller, or larger, shallower or deeper, apparently spontaneous solutions of continuity of the gastric mucosa. In the duodenum also the form of lesion called ulcer is found to have the appearance of a filed-out slit. Dieulafoy describes cases of acute hemorrhagic erosion due to pneumococcal infection of the gastric mucosa and accompanied by copious hematemesis.

Erosions may exist without subjective symptoms but, on the contrary, it has been learned that unexpected acute copious hemorrhage may occur with them. Indeed it seems probable that an acute hematemesis without preceding or subsequent symptoms of ulcer is more often due to erosion than to real peptic ulcer. Toxic hepatitis from focal infection is apparently the underlying cause of such an erosive process in the gastric mucosa.

Many pathologic processes which heretofore have gone unexplained are today accounted for as due to some type of focal infection. There are, however, apparently abundant opportunities for local infection and trauma of the gastric mucosa. Some foods injure the mucous membrane either by their indigestibility or by carrying rather serious septic or toxic substances. Such foods are familiarly known to cause acute gastritis and hepatitis and the pathologic changes incident to a toxic gastritis include erosive lesions. It is, therefore, probable that some cases of erosion occur independently of so-called focal infection which latter presupposes septic embolism or thrombosis of the finer vessels of the mucosa. Whatever their nature and cause these lesions heal without permanent damage and they are thought seldom to develop into true peptic ulcers.

Symptomatology and Diagnosis.—While erosions are usually symptomless from the subjective standpoint their presence may be disclosed by finding superficial fragments of mucosa and blood or occult blood in the stomach contents withdrawn carefully so as to avoid traumatism. Occult blood may be found also in the feces. When, as occurs in a few cases, subjective symptoms are present they consist of a burning gastric discomfort which is at its worst during the more acid digestive period. Slight pain may be complained of or, pylorospasm is usually associated with the irritating lesions, actual hunger-pains may develop. In a hyperesthetic case nausea or vomiting may be present. Eructation and regurgitation may result from the disturbed motor innervation. The gastric contents differ as cases run; in some there is hyperchlorhydria, in others achlorhydria. Mucus, significant of gastritis, may be found. There is no rule regarding the size, position, shape, or motility of the stomach as these characteristics are individual. Nor is there any special radiographic picture though multiple erosions may cause irregularity in the gastric outline, hypermotility, pyloric spasm or tonic contractions of a part of the stomach (gastrospasm).

Erosions may heal in a few days or grow larger, deeper and more irritable. There is no sharp dividing line between local epithelial denudation and the superficial changes marking the beginnings of a small and shallow ulcer. If erosions are deep and destructive enough to cause profuse hemorrhage they run close to ulcer. Are we not possibly dealing with but different grades and degrees of cellular damage?

Treatment.—We are dealing with irritative lesions, solutions of continuity, therefore a soothing diet is indicated.

The medicaments most useful are bismuth subnitrate or subcarbonate in liberal doses, resorcin and icthyol. If there are evidences of gastritis lavage with boracic acid solution, soda or Carlsbad salts solution followed by washing with aqueous fluid extract of hydrastis or mercurochrome serve well. We have used mercurochrome in one-half of one per cent. solution two or three times a week. Weak solutions of silver nitrate are also useful for lavage after cleansing the stomach with distilled water.

If high acid figures are found in the gastric contents, magnesium superoxol is an excellent antacid for combination with the bismuth, or milk and magnesia may be given.

The principles governing the treatment of ulcer are applicable in the management of gastric erosions.

Pathologic Changes.—These vary with the nature of the erosion. Dieulafoy's cases above mentioned, showed a rapid necrosis of small portions of the mucous membrane of the stomach, which occurred during the course of lobar pneumonia, were caused by the pneumococci and were a part of the morbid changes of a general pneumococcia. These illustrate the acute septic type, but in less acute cases, in addition to necrotic changes in the mucous layer, there is time for more inflammatory reaction to take place with an embryonic infiltration and proliferation of the interglandular connective tissue. Erosions are often multiple and are associated with a widespread gastritis. They may heal and recur, perhaps many times, just as may the lesions of cancrum oris. Micro-organisms are found infiltrating the edges of the erosions and also the interglandular connective tissue.

Cruveilhier described two forms of erosion, the follicular and the hemorrhagic and Hayem has given a complete account of both these varieties. In the *follicular type,* the lesion is grayish in color and shows little blood pigment. It may be minute or a few millimeters in diameter. If large enough it has a cut-out appearance with sharply defined edges.

In the *hemorrhagic type* the lesion is dark, hemorrhagic and necrotic. Hemorrhage occurs with this variety. Hayem regards this type as the starting point of eroding ulcer.

GASTRIC AND DUODENAL HEMORRHAGE

Gastric and Duodenal Ulcer.—Gastrorrhagia occurs in about twenty-five per cent. of cases with gastric ulcer. Small hemorrhages occur in many cases without hematemesis. Not infrequently a visible amount of blood is washed from the stomach during the examination of patients who give no history of vomiting blood nor of tarry stools. Blood frequently oozes into the stomach in such small quantities that it is detected only by chemical tests in the form known as occult blood. A sudden copious gastrorrhagia occurs when a minute arterial twig is eroded and opened by an ulcerative process or when a larger arteriole or artery in a state of atherosclerosis ruptures. In the former place sharp bleeding takes place but is ordinarily quenched with a fair degree of promptness, whereas in the latter condition the sclerotic vessel usually spurts and occasions a prolonged uncontrollable hemorrhage as clot formation in the broken artery cannot be readily accomplished. Such hemorrhages as these are very likely to fill the stomach with arterial blood and to cause sudden faintness, distressing nausea, pallor, sweating, rapid, weak heart action and vomiting. The sight of a large amount of blood issuing from the mouth terrorizes the patient at once and the blood pressure may suddenly drop many points. This fear and horror are shown in the countenance of the patient. If a large vessel has been opened and arteriosclerosis with possibly a high blood pressure are coincident, hemorrhage is apt to reduce the red blood cell count below 2,000,000 and the hemoglobin below 40 per cent.; indeed hemorrhage may continue so long and so severe that the patient becomes dangerously exsanguinated. These are, of course, illustrations of acute hemorrhage and may be either gastric or duodenal in origin. Acute duodenal hemorrhage is usually indicated by nausea, faintness and copious bloody stools. Sometimes hematemesis accompanies hemorrhage from the bowel in duodenal ulcer; especially is that apt to be the case if the ulcer is in the first portion of the duodenum. Bloody stools are found associated with gastrorrhagia also but most of the blood is usually vomited, whereas, with duodenal hemorrhage most of the blood is passed by the bowel.

An ulcer may perforate and bleed simultaneously and blood may be found in both peritoneal cavity and the stomach. Hemorrhage is sometimes venous in origin rather than arterial. An ulcerative process may erode a vein of sufficient size to cause a free flow of blood though such a flow is less rapid and copious and more readily checked by natural processes than is that from a spurting artery.

Varicose Conditions of the Gastric Veins.—A form of gastrorrhagia which is grave and in some cases fatal is caused by a rupture of a varicose vein in the gastric wall due to hepatic cirrhosis of the fibrous or biliary types. True varicosities of the gastric veins are very rare. Passive congestion of the mucous membrane of the stomach which is dependent upon decompensated heart disease may also cause venous hemorrhage, but it is not usually severe. Pulmonary emphysema or diffuse pulmonary fibrosis or chronic pleural disease may also give rise to hepatic and gastro-intestinal congestion in which dilated venules may rupture and bleed.

Gastric Syphilis.—Gastric syphilis is sometimes a cause of gastrorrhagia which may result from syphilitic ulceration, a bleeding gumma, specific arterial disease or an associated syphilis of the liver.

Gastric or Duodenal Erosions.—Surgical attempts to check severe gastrorrhagia have demonstrated that it is not always due to well-defined ulcer but to erosions which are found to be either deep, slit-like or superficial. Toxic hepatitis is found associated with erosion in some instances and the pathologic state is supposed to be due to some focal infection such as abscessed teeth, diseased tonsils or paranasal sinuses, appendicitis, cholecystitis, pyelitis, prostatitis, etc. The unheralded sudden onset of hematemesis in young women which was formerly always thought due to gastric ulcer has been found to depend upon erosive conditions of the mucosa above described. Many points may bleed simultaneously in a congestive gastric mucosa. Dieulafoy describes cases having profuse hematemesis from pneumococcal erosions during the course of pneumonia.

Gastric Carcinoma.—Some cases of gastric carcinoma run their course with little or no hemorrhage. Colloid or medullary carcinomata are prone to ulcerate and bleed. Large hemorrhages may take place but slow and continuous or oft repeated oozing of the blood is the common type. The more vascular neoplasms are, of course, those prone to bleed. Scirrhous adeno-carcinoma, the type usually found in the stomach, seldom bleeds profusely. When bleeding takes place in malignant disease of the stomach the contents appear dark, small or large in amount depending upon the quantity of food present and the emptying power of the stomach. As cancer most frequently involves the prepyloric and pyloric portions of the stomach obstructive stagnation of gastric contents exists in most of the cases. Gastric contents vomited or withdrawn through a stomach tube contains small or quite large dark blood clots amid other detritus. Sometimes fragments of a soft neoplasm are found in the gastric contents.

Malignant Disease of the Pancreas.—This may involve and break through into the duodenum causing fatal terminal hemorrhage as was found in one of our cases in the Buffalo General Hospital, or the stomach may be thus secondarily involved. Cancer of the colon may also implicate the stomach or duodenum and similarly cause hemorrhage. Aortic aneurism may rupture into the stomach and thereby cause terrible hemorrhage through that organ.

Parasites.—Ankylostoma duodenale may cause duodenal hemorrhage though the parasite itself usually absorbs the blood. The extreme degree of anemia which characterizes cases of ankylostomiasis results from continuous small hemorrhages and also from the blood absorbed by the parasite and probably by a deleterious effect of some toxin liberated by the parasite.

Foreign Bodies.—Sharp-edged, pointed or jagged foreign bodies may lodge in the stomach or duodenum and cause more or less hemorrhage by traumatizing the mucosa. Astounding numbers of screws, nails, tacks, pins, needles, pieces of sharp steel, bits of glass, etc., have been found in the stomach causing only insignificant hemorrhage and on removal of the offending bodies the mucous membrane has rapidly healed.

Extra-gastric Causes of Gastric or Duodenal Hemorrhage.—The complex and poorly understood blood dyscrasias constitute the underlying etiologic factor in some cases in which hemorrhage either from the stomach or duodenum supervenes.

Chronic hemolytic anemia of the Addison-Biermer Type, pernicious anemia, is rarely accompanied by severe gastro-intestinal hemorrhage. Hematemesis is less apt to happen than is occult bleeding, discovered through examination of the feces.

Splenic anemia on the contrary is a disease which is commonly accompanied by gastrorrhagia and hematemesis and I. P. Lyon calls attention to the danger of mistaking such a hemorrhage for that of gastric ulcer. Gastric hemorrhage, as Osler has shown, may recur over a period of ten years.

Thrombosis of the splenic vein is also accompanied by recurring gastric hemorrhages, resembling Banti's disease in that respect.

Hemolytic jaundice may occasion more or less gastro-intestinal hemorrhage. The spleen is somewhat enlarged. The blood serum, urine, duodenal contents and feces show the presence of uobilin. Bleeding gums, epistaxis and sometimes slight purpura are noted. More or less anemia develops in these interesting, though comparatively rare cases.

Henoch's purpura is sometimes accompanied by slight gastro-intestinal hemorrhage.

Purpura hemorrhagica is associated with very low platelet counts and thrombopenia, with nonretracting clot and great prolongation of the bleeding time. While hemorrhage from the mucous membrane of the stomach and duodenum is not usually severe in this malady yet its recurrence and persistence together with hemorrhages from other parts sometimes induces a severe anemia.

Scurvy and *pellagra* are quite regularly associated with gastric or duodenal hemorrhage. It supervenes as a late symptom and the diagnostic characteristics of these diseases render their detection a comparatively simple matter.

Syphilis hemorrhagica neonatorum and *morbus maculosus neonatorum* may be the occasion of more or less gastro-intestinal hemorrhage.

Acute lymphatic leukemia is fraught with pronounced gastro-intestinal hemorrhage in some cases. Giant purpura, bleeding from the nose and gums with hematuria may be seen. In a case with acute onset giving no history of purpura nor of a previous hemorrhagic tendency leukemia should be suspected. The blood examination will usually render the diagnosis apparent. During the course of the chronic leukemias a hemorrhagic tendency may develop.

Tuberculosis of the gastro-intestinal tract which is often associated with tuberculosis of the mesenteric lymph nodes and the peritoneum may be accompanied by hemorrhage having its origin in the stomach or duodenum, though serious bleeding from this cause is of rare occurrence. Severe acute gastritis, especially if it be of the phlegmonous or necrotic erosive type may give rise to more or less gastric hemorrhage.

Appendicitis is occasionally complicated by hematemesis the immediate cause of which is to be found in hemorrhage erosions resulting from appendicular toxins. Dieulafoy describes several cases and gives the condition the name appendicular vomito negro.

Hanot's disease and *Weil's disease* are conditions which may be attended by gastric or duodenal hemorrhages which is brought about by a morbid hemorrhagic blood state or by passive congestion or erosive inflammation of the mucosa.

Malaria, yellow fever, acute yellow atrophy of the liver, small-pox, measles and *streptococcemias* are infections which during their course may be attended by more or less gastro-intestinal hemorrhage.

Phosphorus, arsenic, bichloride of mercury poisoning and, indeed, any corrosive such as lye or ammonia may so damage the gastro-duodenal mucosa as to cause some measure of hemorrhage. Phosphorus especially damage the liver and also gives rise to hemolysis.

Treatment.—The proper method of dealing with acute hemorrhage from gastric or duodenal ulcer is to **enforce** as **complete rest** as possible, rest of the body and mind, of the stomach and duodenum and of the circulation.

Morphin hypodermatically when well-borne affords ease of mind and, aside from its tendency to allow pyloric spasm, acts favorably upon the bleeding organ and upon the circulation. **Atropin, 1-100 grain,** is often combined with morphin. No food should be allowed and it is best to **deny water by the mouth** but it should be given per rectum by the Murphy drip as freely as the patient is able to take it in that manner. Normal salt solution may be used by the rectum or by hypodermoclysis. If no grave symptoms of exsanguination are noted these measures will suffice if the hemorrhage checks but if not and the pulse grows small and very frequent, pallor, a pinched facies and cold perspiration, air-hunger, ringing in the ears, vertigo and faintness supervene, then **transfusion** by the direct syringe (Lindemann) method is a most excellent procedure as it not only restores the patient but often checks the hemorrhage and may be influential in preventing its recurrence. A light ice bag or Leiter coil may be applied over the epigastrium. It is well to order its removal for an hour after it has been in place for three hours. With a profuse hemorrhage it is useless to give **adrenalin** or **suprarenal extract** by mouth as adrenalin hypodermatically raises blood-pressure, sometimes induces nervousness and nausea and is apt to do more harm than good. It may do good and rarely does harm to use coagulose, thromboplastin, coagulen, or blood serum by the hypodermic method. Small hemorrhages from the stomach may possibly be checked by the oral administration of **suprarenal extract**, one grain, rubbed up well with bismuth subnitrate, 15 grains (.972 gram) every hour or two for four or five doses.

When dealing with bleeding from a crater ulcer which cannot heal, but temporary and unsatisfactory results attend medical measures and excision of such an ulcer is its best treatment. The hemorrhage from cancer may be lessened by **lavage** with **styptic** solutions such as **hydrastis,**

tannic acid, nitrate of silver, adrenalin, coagulose, etc., or these substances may be given orally.

Hemorrhage due to venous stasis demands much the same treatment as does that due to ulcer or hemorrhagic erosions with attention directed to its cause. If hemorrhage accompanies a serious gastritis, the treatment should include starvation, rest, large doses of **bismuth subgallate** and other antiseptic and antiphlogistic measures. If sharp-edged foreign bodies are found to be exciting hemorrhage their removal is, of course, demanded.

The blood dyscrasias must be treated by the best means at our command. Fortunately splenectomy has been found to cure splenic anemia and the same measure is to be employed in the rare cases of thrombosis of the splenic vein if it should be met with.

The control of purpura hemorrhagica has always been most difficult or impossible but more hope comes with the report of cure by splenectomy and one of my ward cases has recently shown a great increase in his platelets following splenectomy. It appears that destruction of the blood platelets takes place in the spleen which is enlarged in this disease in a considerable proportion of the cases and that the hemorrhagic tendency ceases as soon as the splenic artery is tied off. Several cases have recently been successfully operated upon in Europe.

If corrosives have been swallowed **lavage** and **neutralizing solutions** and **soothing demulcents** with hemostatic remedies should be employed.

Scurvy and pellagra call for dietetic and other appropriate treatment while soothing astringents such as **bismuth subgallate, hydrastin hydrochlorate** or **stypticin** may be given. If intense gastric irritability is present anesthesin may be combined with the bismuth.

Prognosis.—The prognosis in patients who suffer from gastric or duodenal hemorrhage depends upon its cause. While the acute gastrorrhagia of hemorrhagic erosions or ulcer is seldom fatal its continuance or repetition may prove a grave affair as the anemia thus induced may be extreme. The hemorrhage from cancer is not often copious and is not in itself a serious matter but the prognosis is bad because of its cause. Hemorrhage dependent upon venous congestion of the gastric mucosa in hepatic cirrhosis is sometimes followed by a prolonged period of comparative well-being but, on the contrary, is in some cases immoderate, exsanguinating and proves a terminal event. Gastrorrhagia resulting from portal thrombosis is always of grave prognostic significance. A free hemorrhage arising from involvement of the stomach or duodenum in cancer of the pancreas or other contiguous structure is of serious moment and is likely to prove of terminat event.

If the mucosa of the upper gastro-intestinal tract oozes blood in purpura hemorrhagica, leukemia or other blood dyscrasia the amount lost is usually not great though its persistence may render the prognosis more immediately unfavorable. The outlook in acute yellow atrophy of the liver is hopeless and that of yellow fever very grave, while that of malaria occurring rarely is not so serious. Hematemesis in infectious or septic processes is apt to accompany a high mortality as it indicates a serious disorganization of the blood and capillaries.

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CHAPTER XV

ACUTE AND CHRONIC DUODENITIS. DIVERTICULA OF THE DUODENUM

By Allen A. Jones, M.D.

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ACUTE DUODENITIS

Etiology.—During the course of some acute infectious fevers acute duodenitis not infrequently develops. It is caused directly by the etiologic agent of the disease, such, for instance, as typhoid fever, smallpox and measles or it results from the toxemia of the disease. During the course of these and other infections, including pneumonia, more or less catarrhal jaundice may appear associated perhaps in some cases with a measure of so-called hematogenous jaundice.

Another etiologic factor of importance is to be found in direct infection of the duodenum with an infective gastritis. The stomach may fail to withstand the onslaught of myriads of pathogenic streptococci reaching it through the esophagus, as in badly infected and diseased tonsils, and its mucosa becomes infected. The duodenal mucosa is later invaded. Or the infection from the tonsils or from abscessed teeth or paranasal sinuses may also cause duodenitis by the hematogenous route. It is probable that both direct and indirect infections are simultaneously operative.

Dietetic causes, spoiled foods and unripe fruit, etc., are also to blame for many cases of acute duodenitis as it probably accompanies gastritis in many cases and is a part of an acute gastro-enteritis.

The most commonly recognized form of duodenitis is that we speak of as catarrhal jaundice, the bacterial or infective nature of which has long been recognized but the exact cause has not been satisfactorily determined.

Studies of the duodenal contents have of late years given us a better knowledge of the bacteriology of that portion of the intestine both in health and in disease. We frequently find streptococci, staphylococci, pneumococci and colon bacilli in the duodenal contents. In some cases typhoid bacilli are discovered and true diphtheria bacilli or tubercle bacilli may be found. Many of these are probably accidental while others are associated with their respective diseases or are discovered some time afterward.

The gall-bladder may harbor some of these morbific agents and they are withdrawn from the duodenum after having passed through the bile-ducts.

Symptomatology.—The symptoms of acute duodenitis no doubt often escape our notice if the affection comes along as a complication of, or is merely symptomatic of, some acute infection like typhoid fever. If, however, the gastro-intestinal symptoms are watched closely during the course of a general infection some of the significance may often be detected. Extreme gastric irritability, a very heavily coated tongue, absolute anorexia and slight icterus with bile-stained urine signify duodenitis and perhaps some degree of cholangitis and cholecystitis. In some cases pain and tenderness in the region of the gall-bladder are present. In case the affection supervenes without an association with another disease the symptoms are those of nausea, perhaps vomiting, coated tongue, anorexia, depression of spirits, headache, constipation and slight rise of temperature. In some cases a chill of undetermined causation ushers in the attack and may be followed by other chills and irregular temperature and sweats. We attribute these symptoms to an involvement of the gall-bladder. Jaundice of some degree is apt to appear two or three days later and we have then the familiar clinical picture of catarrhal jaundice of gastro-duodenal catarrh. How far up the common bile-duct the inflammation must extend in order to cause jaundice is a problem we cannot answer in any given case unless it should happen to come to autopsy from some other cause. Inflammatory swelling at the papilla of Vater is sufficient to cause jaundice, but we probably have much greater extension and involvement than that and there are good reasons for believing that many cases are associated with a considerable involvement of the hepatic duct and of the biliary radicals in the liver with hepatitis. It is probable that some cases at the outset have a hematogenous hepatitis as the liver shows enlargement and some tenderness and dull pain are present. Furthermore, the protracted course of the disease observed in a considerable number of cases suggests a more extensive pathology than a mere temporary swelling of the mucosa at the papilla of Vater. The jaundice assumes a deep hue, is complete and lasts several weeks in such cases and irregular moderate elevations of temperature with a slight leukocytosis may extend over two or three weeks. Slight albuminuria and hyaline and granular casts are very commonly found. The spleen may show slight increase in size.

There is no sharp dividing line between the mild and short cases and the more protracted and severe. With severe symptoms the latter are termed infectious jaundice or Weil's disease. Epidemic jaundice is sometimes observed and the great majority of the cases are apparently of a more or less severe so-called catarrhal type. Patients may begin with mild symptoms and later develop severe manifestations or the case may eventuate in acute yellow atrophy of the liver. These considerations should warn us not to disregard clinical evidences of acute catarrhal duodenitis.

Diagnosis.—In addition to the above-mentioned symptoms of the affection the examination of the duodenal contents shows too many leukocytes and perhaps some red blood-cells and an abnormal amount of mucus which may appear diffuse and hemogenous, giving the contents a mucilaginous consistency, or in the form of small flakes which float in the fluid. The bile may be turbid and dark colored and many micro-organisms are found. Streptococci and colon bacilli are commonly encountered but bacillus proteus, or the other bacteria before mentioned, may be found. Unless there is a very decided swelling and obstruction in the common duct the withdrawal of the duodenal contents by the Meltzer-Lyon method, before and after the instillation of 100 c.c. of 25 per cent. magnesium sulphate solution into the duodenum, brings forth the usual color changes plus the above-mentioned evidences of an inflammatory state of the duodenal mucous membrane. If very turbid bile mixed with much mucus and showing many cholesterine and calcium bilirubin crystals and leukocytes is withdrawn after the introduction of the magnesium sulphate, B. B. Vincent Lyon regards such findings as indicating cholecystitis and cholelithiasis. Einhorn and others conclude that the dark bile comes from the liver and not necessarily from the gall-bladder.

The phenol tetrachlorphthalein test shows delay in the appearance of the dye in the duodenal drippings in case the biliary obstruction is positive and obstinate; also if there exists hepatic cholangitis.

The disease must be differentiated from obstructive jaundice arising from some cause other than duodenitis and cholangitis, from syphilis, and from poisoning by coal tar products, chloroform, phosphorus, arsenic, mushrooms, ptomaines, etc., and from acute yellow atrophy of the liver.

Treatment.—From whatever cause duodenitis arises there are certain principles which apply to its treatment in all cases. The **diet should be designed to give as much rest as possible to the inflamed part and to the liver.** For many years milk has been advised and used by most physicians. For the first two days it is a sensible plan to give only consommé, clear chicken broth, clam bouillon, oyster soup and thin gruels. Then add peptonized certified milk, or milk and Celestin Vichy, or milk and lime water. Later as improvement is evident, zweiback, toast, crisp rolls, milk toast, cereals, custards, cottage cheese and lactis Bulgaricus may be allowed and a simple, easily digested diet of sufficient caloric value may be resumed upon the disappearance of the evidences of inflammation in the duodenal contents and of the jaundice.

Neither gastric nor duodenal lavage are indicated, as a rule, in the early stages of an acute case but if the symptoms prove obstinate and threaten chronic duodenal catarrh both should be employed. The best drugs are small divided doses of **calomel** and good-sized doses of **bismuth subnitrate or subcarbonate** or, as a more definite antiseptic, **bismuth salicylate** or **bi-salicylate.** Gastric acidity should be lowered by **magnesium oxide or carbonate.** If there be fever and headache and the stomach is not too irritable, magnesium salicylate, 5 **grains (0.324 gram),** may be combined with the above-mentioned remedies.

Each morning a pint of **hot Carlsbad water,** or a teaspoonful of Sprudel salts dissolved in a pint of hot water, should be sipped slowly a half hour before any nourishment is taken. Sodium phosphate may be substituted for the Carlsbad. The patient should preferably be at rest in bed for the first few days and have a bran and flaxseed poultice, or ammonium muriate solution, hot compress, applied over the upper abdomen every four hours, for one hour, during the day and evening.

CHRONIC DUODENITIS (CHRONIC DUODENAL CATARRH)

One or more attacks of acute duodenitis may eventuate in chronicity and the inflammatory processes thereafter undergo remissions and exacerbations. Chronic duodenal catarrh usually affects the bile-ducts and involves the pancreatic duct. In some cases chronic cholecystitis, hepatic cholangitis and pancreatitis follow extension of the morbid process.

The usual pathologic changes attending chronic inflammation develop in the duodenal mucous membrane and in the biliary passages and pancreas if they become involved. It is not necessary here to review those changes as space will not permit.

Symptomatology.—The symptoms vary greatly in different cases. The condition may exist without giving rise to definite or noticeable symptoms but, on the other hand, there may be persistent digestive complaint. The appetite is apt to be poor and fickle, there is considerable eructation, some regurgitation, periods of nausea are common, distress which may resemble hunger-pain may come on two or three hours after eating, the tongue is heavily coated. There is no rule as to the condition of the bowels, they may be regular, loose or constipated. As the affection is commonly associated with chronic gastric catarrh the food propelled within the duodenum is mixed or coated with mucus and is partially undigested, so that the impaired duodenum is insulted and overtaxed during each digestive period. If the intestine below fails to compensate for the defect above more or less diarrhea may result. Headaches, mental and nervous depression and repeated attacks, characterized by the laity as bilious attacks, mark the existence of the morbid condition. Some anemia may be noted.

If the mucous membrane of Vater's papilla undergoes swelling there develops a sallow pasty complexion or recurrent attacks of slight icterus may be observed. If pancreatitis of a low grade chronic type be brought about, loss of weight, undigested foods in the feces, and perhaps glycosuria may be noticed.

Examination of the gastric and duodenal contents should be made and the stools should also be studied, as much valuable information may thus be gathered both for diagnostic and therapeutic use.

The stomach contents may be overacid, normally acid or below normal in acidity. There may be a pathological amount of gastric mucus, and too many leukocytes and desquamated epithelia may be observed in the contents from the fasting stomach. Of course allowance must be made for the morbid cellular elements entering the stomach from the naso-pharynx and mouth. In some cases achylia gastrica is found.

The duodenal contents show abnormal quantities of mucus in most of the specimens. There are pathologic numbers of leukocytes and epithelial cells; there are usually many microörganisms and if the functions of the pancreas are impaired the amylopsin, steapsin and trypsin values are lowered. Several examinations of the contents should be made on different days in order that the results may be compared and, if the patient is under treatment, progress or retrogression noted. Slow and insufficient flow of bile may follow the instillation of magnesium sulphate, sodium phosphate or sulphate, peptone, glucose or olive oil into the duodenum. Widal's digestive hemoclastic test should be made, as it is a valuable index as to impairment of the function of the liver. Phenoltetrachlorphthalein introduced intravenously, as practiced by Aaron, Beck and Schneider, may show some delay in its appearance in the duodenum. These findings are indicative of some degree of hepatic insufficiency which is to be expected as a late result.

If feasible, x-ray examination of the stomach and duodenum would better be made in order to ascertain whether or not a duodenal diverticulum exists. Radiographic information regarding the stomach (ulcer), pylorus (spasm), duodenum (ulcer) and gall-bladder (stones) may prove valuable.

Treatment.—The excellent and noteworthy results B. B. Vincent Lyon has had in many cases of **duodenal drainage** and **lavage** should encourage us to use this method of treatment in these cases. The reader is referred to his valuable contributions for definite instructions as to this systematic method of duodenal therapy.

If gastritis is present, lavage should be practiced and appropriate diet and remedies given. Prompt relief from headache, anorexia, and nausea will usually follow the taking of **calomel** or **blue mass** followed by a **saline**, but the too free and frequent use of mecurial or other purgatives is to be deprecated. Carlsbad water, Sprudel salts, Pluto water, Hunyadi water, Abilena water, are all excellent if used well diluted and judiciously. If constipation is present a combination of **bile salts** with **phenolphthalein** is useful. Of late years, capsules, tablets or pills containing different combinations of sodium succinate, sodium glycocholate, sodium taurocholate, purified oxgall, salicylic acid, sodium salicylate, menthol, phenolphthalein and cascara have been put upon the drug market and have proven helpful in many cases. **Pepsin, taka-diastase** and **holadin** may be given.

Out-of-door **exercise** such as golf, tennis, canoeing, rowing, horseback riding, and walking are especially to be prescribed, but those who are thin, wan, overtired and weak should be treated if possible in a good sanitarium or hospital at first and should be directed cautiously in their course of life.

Finally, these measures may prove of little avail unless the diet is properly managed. As we are dealing with a chronic inflammatory state of a very important part of the digestive tract simple, digestible, unirritating, nutritious foods should be prescribed:—chicken, oyster and clam broths, purées and consommé, the finer cereals, zweiback, light white and whole wheat flour bread, toast, milk toast, farina, well boiled and steamed rice, soft poached, boiled or scrambled eggs in moderation, fresh white fish, bass, pickerel, pike, perch, brook or lake trout, salmon and shad; white meat of tender chicken, lamb and beef in small amounts; baked and boiled light mealy potatoes, spinach, chard, beet greens if put through a colander or chopped very fine, boiled tender young beets, carrots, parsnips, peas and lima beans and squash; custards baked and boiled; cottage cheese, buttermilk; as desserts fruit jellies, apple sauce and baked apples, orange and grapefruit juice, tapioca, sago and bread puddings, cornstarch, sponge cake, plain cookies not too rich; a little weak tea and coffee clear. Vichy, Contrexeville Arondack, Poland, Bethesda or soft pure water should be obtained if possible.

Mastication should always be thorough, and hurry and worry eliminated at meals.

All rich, fried, too greasy, too sweet, too coarse, bulky and indigestible roughage should be avoided. Alcoholic beverages are decidedly contraindicated.

DUODENAL DIVERTICULUM

Etiology.—Although rare there are now, by radiographic diagnosis, well-defined cases of duodenal diverticulum discovered. The diverticulum may be true or false and is most often at the site of Vater's papilla though it may exist in any portion of the duodenum. In a careful study of the subject Holsweissig states that diverticula near the pylorus are often associated with ulcer and are the result of traction in cicatricial tissue, their walls containing all intestinal layers. Other diverticula consist of the mucous layer only and are to be considered as pulsion protrusions through the musculature where the common duct passes through the intestinal wall or along the course of a blood-vessel. For some reason the tonus of the duodenal wall is impaired and this is accentuated probably by chronic pancreatitis or pancreatic lipomatosis which is generally present. The vessels were found to enter or traverse the wall perpendicularly instead of obliquely and passive congestion, by enlarging the vessels, rendered pulsion of the mucous membrane around it the more easy.

The musculature surrounding the common duct may be split by small fibro-adenomata originating in the mucous glands of the duct, thus lessening its resistance.

Keith regards corset visceroptosis as a cause of diverticulum at the papilla in women upon the explanation that the bile and pancreatic ducts owing to their more fibrous nature do not readily descend with duodenal ptosis and that some pouching at the papilla thereby results. Diverticulum at the duodenojejunal flexure of the extroversion type is reported by Sanlander. Radiologic diagnosis was confirmed by operation. The data upon which diagnosis rests are these: A persistent shadow in the duodenum past which the contrast ba-meal in watery solution, as recommended by Holznecht, is seen fluroscopically to flow, disappearance of this spot-shadow occupies many hours or maybe days, the depot is movable toward the posterior abdominal wall. Freud is said by Herrnheiser to include the presence of peristalsis in the diverticulum but this must be rarely distinguishable. Differentiation should, if possible, be made between an ulcer pocket, ulcer diverticulum, covered perforation, *barium-filled* gall-bladder after cholecystostomy and visible gallstones. Ulcer pocket is suggested by the presence of Handek's air layer. by a fixed position and tenderness on pressure. Duodenal diverticula are sometimes present with multiple diverticula of the rest of the small and of the large intestine.

Symptomatology.—The subjective symptoms of the condition may be very slight or indefinite. There may be some epigastric discomfort or pain and eructation is common. Exercise and the erect position may aggravate the distress while the dorsal position may relieve it. In some cases the ingestion of food is followed regularly by distress or pain; indeed, varied symptoms of chronic digestive disturbance may attend, or result from, the condition. The cases we have seen with duodenal diverticula had rather vague and unexplained digestive symptoms of long standing and during routine study the condition was disclosed by fluoroscopy and radiography.

Whenever a diverticulum exists it may at any time become the seat of diverticulitis and in such a condition jaundice may develop if the pouching is, as most commonly occurs, at the outlet of the ductus communis choledochus. Other symptoms such as nausea, increased local discomfort or pain, local tenderness and slight rise of temperature and leukocytosis may be also present.

Treatment.—If the diagnosis has been previously made and symptoms of an inflammatory state in the diverticulum supervene, the patient should be put to bed as a localized peritonitis or even perforation may occur as in acute diverticulitis of the sigmoid or elsewhere. Duodenal lavage may be practiced with soothing, slightly astringent remedies such as normal saline with oxgall and hydrastin. It is well to be cautious about using drug ablutions in the duodenum as it seems much more sensitive than the stomach.

For the rest if symptoms which seem reasonably certain to depend upon the existence of a duodenal diverticulum prove obstinate and unyielding surgical measures should be adopted.

Radiography.—The radiographic observation of diverticulum of the duodenum is as follows: Diverticulum of the duodenum is not common and when it occurs usually is not from duodenal ulcer but is more apt to be from deformity of the duodenum by pulling of adhesions producing a small sacculation which hold portions of the opaque meal.

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CHAPTER XVI

CANCER OF THE STOMACH, SARCOMA OF THE STOMACH, BENIGN TUMORS OF THE STOMACH, TUBERCULOSIS OF THE STOMACH

By Seymour Basch, M.D.

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Sarcoma of the stomach, p. 506:—Definition, p. 506; Etiology, p. 506; Symptomatology, p. 506; Complications, p. 507; Treatment, p. 507; Prognosis, p. 507; Pathology, p. 507.
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CANCER OF THE STOMACH

Definition.—Cancer of the stomach, or carcinoma ventriculi, is a neoplasm of a primary or secondary type, which has its beginning in the glandular structures and subsequently invades and destroys other tissues of the stomach.

Etiology.—Although many theories have been advanced to account for the disease, none has found general acceptance. That the stomach is the part of the human anatomy which is the seat of cancer in more than half of all persons who are afflicted with this disease, may be explained in two ways:

(1) The Acidity of the Gastric Juices.—Illustrative of the fact that acidity is an important factor in the production of gastric cancer, it is well to mention here that though the duodenum is anatomically a part of the stomach it is seldom the seat of a primary cancer. Moreover, gastric cancer very rarely develops from a chronic ulcer after gastroenterostomy has been performed, since the intestinal secretions which flow back into the stomach are alkaline.

(2) The contracting orifices—the pylorus and cardia. These are frequently exposed to trauma, hence their greater susceptibility to malignancy. That irritation, whether mechanical or chemical, is a causative factor, has been clinically established so often that its acceptance no longer invites criticism. Despite this acceptance we are still in a state of doubt as to whether even repeated irritation can produce cancer in

healthy tissue, whether an embryological cell-rest must first exist, or whether the tumor occurs because of a deficiency of protective substances.

The frequent association of chronic indurated ulcer and cancer has given rise to the thought in the minds of many pathologists and surgeons that a close etiological relationship exists between these two diseases, but it is an indisputable fact that the vast majority of chronic ulcers heal either spontaneously or after medical treatment, and, moreover, fail to show any trace of a cancerous growth at operation.

The fact that the cancer years are between forty and sixty has never been satisfactorily explained; neither has a satisfactory explanation ever been made as to what part heredity plays in causing the disease. There is a racial point that should not be overlooked or treated lightly, namely, that the dark-skinned races of Africa and Latin America enjoy a greater degree of immunity than do the light-skinned races. As soon as the underlying cause of this fact is ascertained there is no doubt that we will be nearer the solution of the cancer problem. Exhaustive study and investigation along the above lines may therefore be advisable.

Symptomatology.—There is no other disease which requires a greater degree of exactitude in the study of the patient's symptoms, or taxes to a greater extent the diagnostic ingenuity of the clinician than does cancer of the stomach in its earliest stages. It should be the invariable rule with all clinicians that when a patient in the cancer years presents himself with a severe gastric complaint, malignancy should receive the first consideration.

Clinical History.—By careful questioning the patient a history of chronic ulcer may be elicited, a condition of which the patient himself may have been entirely unaware—one characterized by periodic repetition of the gastric disturbances, especially sour belching, burning and distress after eating with intervals of normal appetite and perhaps even well-being. On the other hand the patient may recall having had one or more sudden attacks of general weakness with tachycardia or possibly syncope symptoms, suggestive of hemorrhage from a gastric ulcer. When presenting himself for examination the patient has gastric symptoms during the entire twenty-four hours and these symptoms are not so closely connected with the ingestion of food as is the case in gastric ulcer; in other words, though the symptoms characteristic of gastric ulcer still predominate, there are premonitory signs of gastric cancer, easily overlooked by the less acute observer. Increasing aversion to food, particularly to meat, may be present, and progressive loss of weight and vigor noted. In short, the whole physical appearance suggests a marked decline in health.

In those cases where no history of gastric ulcer can be arrived at, but where the gastric complaint has been sudden and acute with no previous indications, patients usually refer their trouble to some dietary indiscretion or some other improbable cause. We find, however, in these cases there are the same increasing anorexia and aversion to meat, with similar dyspeptic symptoms of pressure, gas, belching and even pain, not necessarily following a meal. Vomiting may occur even in the earliest stages, sometimes with the appearance of visible blood streaks which alarm the patient so greatly that he seeks medical aid. A case of this sort, failing as it does to respond to simple customary treatment, and in which physical examination reveals on palpation local tenderness or the signs of infiltration or induration of the stomach, is perhaps the earliest visible picture of a primary carcinoma. This applies more particularly to growths in the body of the stomach. In these growths nearer to or at the orifices there are, in addition, spasms, signs of obstruction or sometimes even of a patulous orifice. The alternate possibilities of sarcoma, a benign tumor and syphilis, should be born in mind and ruled out.

PHYSICAL FINDINGS.—A typical advanced case of gastric cancer is characterized by marked emaciation, extreme weakness and pronounced pallor, even to cachexia. The fatty tissues have disappeared, the muscles become wasted and enfeebled, while the bony structures are very prominent. As a rule the tongue is heavily coated with a dirty grey and brownish fur, the breath foul and gums in poor condition, the state of the mouth depending largely upon the care bestowed upon it. With the occasional exception of Virchow's gland in the left supraclavicular region, it is rare to find any palpable enlarged surface lymphatic glands.

Edema of the legs, as Boas long ago pointed out, is an early manifestation, occurring in about 12 per cent of all cases. The abdomen is sunken and almost always readily palpated. Both inspection and palpation reveal the presence of a more or less extensive tumor mass in the stomach area. This mass is hard and irregular in shape; its surface may be smooth or nodular; the tumor is usually tender to the touch. Depending upon its situation and other associated factors, the tumor may or may not move with respiration and digital palpation. It should, however, be remembered that even a very large and inoperable growth in the so-called "silent area" of the stomach, *i. e.*, behind the left lobe of the liver, may elude most careful palpation even under a general anesthesia. Here the diagnosis must rest with the history, the physical condition of the patient, together with the laboratory and roentgenological findings.

In the sclerosing type of cancer, the walls of the stomach are very much thickened and hardened and the entire organ shrunken in size with an almost obliterated lumen. The stomach, therefore, in these cases presents a very hard tumor mass whose content is small in amount and rapidly passes into the duodenum without detectable peristaltic action. In cancer involving the cardiac opening, the stomach generally is also markedly decreased in size. Tumors at the pyloric end are as a rule associated with mechanical obstruction, stagnation of content and secondary dilatation of the stomach. They evidence, therefore, not only the tumor mass itself, but also the well-known manifestation of gastrectasy, namely, greatly enlarged stomach outlines (inspection and palpation), splashing sounds, gurgling, etc., more or less visible peristaltic over-activity, so-called "peristaltic stiffening," retained stomach contents in the fasting state, vomiting of profuse amounts, etc.

LABORATORY FINDINGS.—*Examination of the Blood.*—Much has been hoped for in the way of discovering something in the blood specific of cancer. Thus far all investigations have proved futile. Recently a more definite promise of diagnostic value has been put forth by Rodenburg, Friedenwald, and others in connection with blood sugar tolerance test. However, this method is too recent to permit of a definite estimate of its real value. Parallel with the progress of the disease there develops a secondary anemia, which in advanced cases is so profound as strongly to suggest the primary pernicious form of anemia.

Urine.—This shows nothing that is pathognomonic of the disease.

Gastric Analysis.—This should be conducted with a view to determining, (*a*) changes in secretory function, and (*b*) changes in motor function.

(*a*) Cancer being a disease that originates in the glandular elements of the stomach and is very soon accompanied by an infiltrating gastritis, there results sooner or later a gradual diminution of the normal gastric secretions and simultaneously a production of catarrhal and cancer products. The hydrochloric and peptic ferments disappear and excessive mucus, lactic acid, Boas-Oppler bacilli and more or less free and occult blood become manifest. Since it has been found that cancer also produces a specific protein digesting ferment, this fact has been utilized as a point of differential diagnosis between malignant and benign achylias. Upon this fact are based the Salomon and Wolff-Junghan's tests. The author has found the latter test to be very valuable and has employed it regularly as a diagnostic aid. It is easily carried out, is inexpensive, and requires but little time. Smithies found it positive in 80 per cent. of 230 cases of operatively or pathologically demonstrated gastric cancer. Friedenwald and Kiefer also found this test most serviceable in the study of cases. The presence of lactic acid and more especially of the Boas-Oppler bacillus has long been recognized as an especially important point in the diagnosis of gastric cancer. In this connection it should be remembered that where cancer supervenes upon chronic ulcer, the latter for a very long time is the more manifest of the two diseases; hence there is a persistence of much free hydrochloric acid, until finally the cancerous nature of the disease predominates. This point should always be born in mind in making a differential diagnosis between cancer and chronic peptic ulcer.

(*b*) Changes in motor function vary according to the parts of the stomach invaded by the growth, and have just been discussed. A more detailed description of pyloric obstruction is given in another part of this volume.

Blood in Stomach Contents.—Where brought forth in large quantities, blood may be macroscopically visible, appearing as free frank blood, or more frequently may have the well-known "coffee ground" appearance, especially when the hemorrhage is slighter and slower and the blood becomes coagulated and has time to intermix with the stomach contents. Blood often appears, however, in minute amounts, invisible to the naked eye and is only demonstrable chemically through the guaiac, benzidin, and other well-known tests. Occult blood in the feces is more frequently found in cancer of the stomach than in ulcer.

ROENTGEN EXAMINATION.—The roentgenological evidence and diagnosis of gastric cancer are fully discussed in the chapter by Dr. F. H. Baetjer (Vol. VII, pp. 241-284). The x-ray is not an infallible means for early diagnosis, but together with the history and the laboratory reports, it usually completes the evidence in the primary type of gastric cancer. But in postulcerous cases differential diagnosis by the x-ray is very difficult in the earliest stages. It is the author's experience that tumors in the body of the stomach may attain even a large size without becoming distinctly visible under the x-ray, and not infrequently the growth is much more extensive than indicated by the roentgenogram. When negative x-ray findings contradict strong clinical and laboratory evidence, the patient should be kept under close supervision and the roentgenographic examination repeated at intervals until the case is cleared up. Positive x-ray findings in early or in doubtful cases always call for exploratory incision.

Differential Diagnosis.—The data above mentioned as associated with a typical advanced case are, taken collectively, so characteristic that, when for the most part present, they make the diagnosis unquestionable. The only conditions closely resembling such an advanced type typical cancer are sarcoma, massive benign tumors and syphilis. The differentiation of these from cancer, is discussed in their respective chapters. Frequently, however, a few or even most of the characteristic data may be absent and it may be impossible short of laparotomy with histological examination of the tumor to establish the correct diagnosis within a brief period of observation.

The great importance of the histological examination cannot be sufficiently emphasized. There is so much at stake concerning the prognosis, the correct operative procedure, the attitude of those closest to the patient and, last but least, the surgeon's reputation, that a histological examination should always be demanded, be the surgeon ever so experienced and skillful. Instances can be multiplied where the operative diagnosis of cancer, based upon the presence of a very hard and large tumor mass, was refuted by the subsequent career of the patient, or by the report of the histologist or serologist. On the other hand, post-mortem examinations and exploratory laparotomies have sufficiently often disproved the presumptive diagnosis made by the internist of an "unquestionable case of cancer of the stomach."

For the purpose of simpler discussion, we may divide the differential diagnosis into cases in which, a palpable tumor mass is absent, and those in which it is present.

CANCER WITHOUT TUMOR.—In the absence of a palpable tumor there will be difficulty in distinguishing between cancer and other gastric conditions associated with marked diminution or complete absence of normal gastric juice, viz: chronic gastritis (simple, hemorrhagic and syphilitic); benign (functional) achylia gastrica and pernicious anemia.

Differentiation from Chronic Gastritis.—The fact that a catarrhal state almost always accompanies cancer, may at first give rise to some speculation when one encounters a gastritis in an individual within the cancer age. In both diseases test meal examination reveals excessive mucus and diminished gastric acidity. Here the present, and more so the previous histories are of prime importance. There is the same lack of appetite, especially the aversion to meat, similar dyspeptic complaints (gas pressure, bloating, belching and nausea). As a rule these last are constant in cancer and have only recently evidenced themselves, whereas in gastritis they are of long duration and follow only upon or are more pronounced after the ingestion of food.

In cancer there is, too, a stronger suggestion of a serious constitutional disease. The point tenderness in the latter affection is local, while in benign gastritis, the tenderness, if present at all, is diffuse over more or less of the gastric area. Blood is present, too, in hemorrhagic gastritis and hence the differentiation is much more difficult. In the early stages it may in fact be impossible. Later still the differentiation becomes more decided, owing to the development in cancer of a tumor or a local resistance with tenderness, of a Wolff-Junghan's reaction, of lactic acid, blood, etc. The x-ray findings are of course essentially different and should readily clear up the differential diagnosis.

Benign (Functional) Achylia Gastrica.—This is differentiated from cancer by a longer history, better physical well-being, absence of real pain and better appetite. There is a tendency to diarrhea and other intestinal disturbances in benign achylia. In cancer vomiting is common; in benign achylia rare. The tongue is coated, the breath foul and the gums usually in poor condition in cancer; in benign achylia gastrica the mouth conditions are normal. As a rule the stomach content and feces in benign achylia lack the presence of blood so characteristic of cancer; lactic acid, too, is absent and the Wolff-Junghan's reaction decidedly negative. Tumor formation, resistance or decided point tenderness are not associated with simple benign achylia gastrica. Finally, the x-ray findings are positive and as a rule diagnostic in cancer, while they are negative in benign achylia gastrica.

Pernicious Anemia.—When a characteristic cancer tumor is present, the diagnosis is very evident. But in the absence of a palpable tumor, the differential diagnosis, especially from an early cancer, at times may be most difficult or even impossible. With a characteristic blood-picture of pernicious anemia on the one hand, or a positive Roentgenogram of cancer on the other, the diagnosis is easily cleared up. But frequently these findings are atypical and the problem becomes more difficult. For while the anemia in cancer, even when profound, is always of a secondary type, definite criteria of primary anemia at times are lacking in cases of pernicious variety. The writer has encountered cases where only the autopsy or exploratory laparotomy gave the correct solution. Cancer cases with very few exceptions markedly and progressively emaciate, while in pernicious anemia even to *exitus* the patient usually remains relatively stout. In pernicious anemia, too, a complete achylia of the benign type is almost invariably present, and what has been mentioned under benign achylia in regard to the differentiation of the stomach content applies equally well here. The presence of any diagnostic tumor fragments in the vomitus or the stomach content at once clears up the diagnosis.

Cancer with Tumor.—The presence of a tumor, definitely proven to be gastric, is the most important physical sign of cancer. When associated with marked emaciation cachexia and upon the characteristic laboratory and x-ray findings, the diagnosis becomes almost a positive one. It should be born in mind, however, that any variety of intragastric tumor and also some extragastric ones that cause pressure upon the stomach, may give rise to almost identical physical phenomena, so that a differential diagnosis may be extremely difficult or even impossible, without resorting to exploratory incision or autopsy. Even in the last, eventually we may have to await histological determination.

The author has found repeated and carefully carried out study of the gastric contents to be of greatest value in the differentiation of gastric cancer from other intra- and extra-gastric tumors. While it is true that all the so-called specific criteria for gastric cancer from which so much was hoped for have definitely failed, we must remember that many of them possess diagnostic values of relatively high degree. Thus, the presence of lactic acid, the finding of strongly positive reactions for cancer ferment (Wolff-Junghan's, Solomon's, Gluzincki's, etc., test), occult blood in the stomach contents and feces, etc., strongly favor the probably presence of a gastric cancer. It is obviously impossible, where proper laboratory facilities are not readily available, and indeed unnecessary, even when these are at hand, to carry out even a majority of these specific tests. The author has almost entirely discontinued tests carried out with difficulty and finds that sufficient diagnostic aid in the average case can be obtained through careful observation of the gross appearance of the stomach contents. Ordinary microscopic examination and simple chemical tests. The necessary details have already been discussed under laboratory findings. Lactic acid is the best known chemical characteristic of cancer, but the author regards a positive Wolff-Junghan's reaction as the most dependable sign in the differentiation of a malignant achylia from a benign one.

Extragastric conditions presenting tumors simulating cancer of the stomach include cancer of the liver, pancreas, duodenum, peritoneum and mesentery, enlarged lymph glands and aneurysm of the abdominal aorta. The limitations of space prevent any detailed discussion of their differential diagnosis.

In most instances the location and general characteristics of the tumor, together with all the important facts deduced from the history, physical, laboratory and especially the x-ray examination will enable us to readily decide the intra- and extra-gastric nature of a tumor. Naturally, a gastric tumor will be restricted in its location to the confines of the stomach itself. This organ, as we have learned from roentgenology, presents the greatest variations in position, size, shape and mobility.

That fact should always be borne in mind when we meet a palpable tumor of the upper or mid, and occasionally, too, of the lower abdomen. The gastric region involved by the growth largely determines its location in the abdomen. Thus, pyloric tumors lie to the right of the midepigastrium, usually just below or even behind the liver, and are therefore only partially or not at all palpable. Unless adherent to the liver, they do not move with respiration. If, however, the stomach is ptosed, they, too, may come to lie quite low down and be palpable in their entire extent. Tumors of the greater curvature and of the body of the stomach are found within the median area of the abdomen, above or below the umbilicus. They are readily palpable unless limited to the posterior wall, in which case they are usually entirely inaccessible to the touch, especially in a filled state of the stomach. In the fasting stomach, tumors of the posterior wall, may rarely manifest themselves as an indefinite resistance. Since the cardiac half of the lesser curvature and the upper portion of the body of the stomach lie high up behind the left lobe of the liver, tumors limited to these parts are almost entirely inaccessible to palpation excepting when very large, or in conditions of marked stomach ptosis. When felt at all, it is only upon very deep inspiration and then high up behind the left costal arch. Growths at or near the cardia are never accessible to palpation.

Information of more or less value for the correct localization of a gastric or extragastric tumor may be gained through inflation of the stomach with air or carbon dioxide. This method, according to Boas, causes the stomach to rotate upon its long axis, the greater curvature passing forwards and the lesser one backwards. Thus, pyloric tumors move further to the right and upwards or downwards; those of the lesser curvature move more deeply backwards into the abdominal cavity, while those of the greater curvature pass forward. These last, as well as those on the anterior surface of the stomach, then seem larger than before, although their borders are less distinct. Tumors of the liver are pushed upwards and towards the right, the edge of the liver becoming more palpable. Pancreatic growths, enlarged peritoneal glands and other tumors lying behind the stomach disappear entirely; Splenic tumors, unless fixed through adhesions, are pushed to the left or downwards and those of the transverse colon and of the omentum pass downwards.

In the average case a detailed study utilizing the diagnostic criteria, as above elaborated, pertaining to the history, the physical and laboratory findings and wherever possible, combined with a competent roentgenological examination, will readily suffice to establish a definite diagnosis for or against the presence of a gastric cancer. Occasionally an extremely atypical case will be encountered, that will baffle the skill of the most experienced and expert investigator, and a correct diagnosis will be impossible short of surgical and perhaps, too, of histological investigation. Between these extreme types one not infrequently meets with a case that puzzles the average investigator. Here clarification of the situation can be arrived at only through most painstaking attention to every diagnostic detail, coupled with intelligent application of past experience, bearing well in mind all conditions that may possibly fit into the clinical picture.

Complications.—Aside from the usual migration through the lymph channels and less frequently through the blood to neighboring and remote parts, there follow adhesions to or direct extensions into the liver, pancreas, gall-bladder and other adjacent viscera. More rare are massive hemorrhages and acute or slow perforations into the peritoneal or pleural cavities, with formation of perigastric abscess, sulphrenic abscess and pyohemothorax.

Treatment.—CURATIVE.—All other methods of treatment including the specifics having proved futile, our only hope of prolonging life and effecting cure is in gastrectomy. All authorities insist upon early operation, but the postoperative statistics will improve greatly when surgery is more often resorted to before the diagnosis has attained a fatal clearness. To await for absolute certainty till we are confronted by a malignant tumor may occasionally avert an unnecessary operation, but it unfortunately works havoc on the side of sacrificing possible salvage. After we have the classical signs of cancer—a palpable tumor, obstruction, food remnants, deformities or muscular deficiencies revealed by the radiographer, Boas-Oppler bacilli—the surgeon generally finds a hopeless state of affairs because of extensive metastasis, adhesions to inoperative organs, the patient's inability to withstand the shock and the like. The outcome is of course always dependent upon the variety, situation and extent of the tumor, but since the only alternative is a mortality of 100 per cent. the patient has nothing to lose by prompt operation at the very earliest moment that a *reasonable suspicion* of carcinoma is entertained. Then there is at least a chance that surgical skill can make a wide removal which includes every trace of cancerous tissue.

Recently deep x-ray therapy has been advocated for the prevention of recurrence in radically treated cases through the destruction of possibly unremoved malignant cells. This procedure while very promising must be regarded as still *sub judice.*

PALLIATIVE.—In advanced cases with cardiac obstruction, gastrostomy, with subsequent direct feeding may require consideration and in pyloric obstruction possible gastroenterostomy. But since patients with advanced cancer stand surgical shocks very poorly, it is usually better not to subject them to the hazard of operation. The author's experience is that these hopeless cases live longer and more comfortably under a strictly medical treatment consisting of cheerful surroundings, proper diet, gastric lavage and symptomatic medication. While the physician must of course tell some member of the family what the ailment is, it is unjustifiable cruelty to let the patient know the truth about it. The sufferer should be cheered up by an optimistic diagnosis, prognosis and explanation of all his symptoms, and should be encouraged to go about according to his past habits so long as his condition permits. When there is no mechanical interference with the passage of food, the diet should be as liberal as the appetite and the powers of assimilation allow. In early cancers, either primary or postulcerous, the appetite is frequently surprisingly good. Usually the diet should be liquid or semisolid; meats and notoriously indigestible foods as well as overeating should be avoided; mild condiments and dainties to stimulate appetite are useful. In very advanced cases it may be necessary to supplement nutrition with rectal alimentation. Experience has taught the author to abandon the old-time food enemas, as they are poorly absorbed and make the patient uncomfortable. Plain saline, sodium bicarbonate or glucose (4 or 5 per cent.) solutions by the Murphy drip method, or whiskey in doses of 1 to 5 ounces of water, injected slowly into the rectum, are decidedly preferable. Topical application of fomentations, the hot water bag or the electric pad, have a soothing effect.

Gastric lavage is indicated primarily in pyloric obstruction. It is also useful where marked accumulation of mucus in the stomach interferes with appetite and digestion. For this purpose saline or mild alkaline solutions are employed, but lavage should not be done daily as it tends to exhaust the patient. The appearance of free blood either spontaneously or upon lavage is a definite counter indication to further lavage.

The medicinal treatment should be symptomatic. The so-called "stomachics" the time-honored "vegetable bitters" coudurango, cinchona, etc., as well as drugs such as orexin, orthoroform, valerian, cerium oxalate and ethereal spirits have proved absolutely worthless. Short of opiates the pain is best relieved by anesthesin, chloroform water and validol. The following prescriptions are frequently useful:

Anesthesini, grains i-ii (.065-.130 gram).
Cerii oxalatis, grains v (.324 gram).
Bismuthi subnitratis, grains x (.65 gram).
M. Ft. pulv. No. 1. One every 3 or 4 hours as required.

Or

Cocainæ hydrochloratis, grain ⅓ (.0216 gram).
Bismuthi subnitratis, grains x (.65 gram).
Aquæ chloroformis, *f* ʒ i (3.75 c.c.).
M. Shake. Take every three hours until relieved.

Dilute hydrochloric or phosphoric acid (5 to 10 minutes before or after meals) may help to improve the appetite. When the suffering becomes intense, which may be quite early in the disease, the resort to opiates should not be delayed to the point of heartlessness. Their disadvantage of decreasing appetite, increasing exhaustion and clouding mentality is offset by the saving in wear and tear on the patient, the relief from intolerable pain by quiet sleep, and by the mental euphoria.

One should, of course, begin with the smallest effective dose and increase it only as the patient's needs demand.

By mouth, morphin sulphate may be given in from 1/8 to 1/2 grain (.0081 to .0324 gram) doses, or deodorized tincture of opium in doses from 5 to 10 minims (.3 to .6 c.c.); also codein sulphate or phosphate in doses of from 1/4 to 2 grains (.0162 to .13 gram). Oral administration should, however, be reserved for cases without marked vomiting or pyloric obstruction.

Per rectum, suppositories of codein, 1/2 to 2 grains (.0324 to .13 gram), plain or with the addition of 5 grains (.324 gram) of aspirin, seem to act better than morphin. The extract of opium (1/2 to 2 grains), with or without the addition of extract of belladonna (1/3 to 1/2 grains, .0216 to .0324 gram), is also frequently employed in suppository form.

Hypodermatically, morphin may be given (1/6 to 1 grain, or even more in emergency conditions), or codein sulphate or phosphate (1/4 to 2 grains).

Prognosis.—Despite numerous claims from various sources, the accepted opinion of the medical profession to-day is that gastric cancer is curable alone through surgical means, and then only under most favorable conditions of location and extent. Cases of spontaneous recovery are occasionally mentioned in the literature, but they lack authentic verification of diagnosis. Naturally, the earlier the surgical procedure, the better the chance for radical removal and cure. Tumors at or near the pylorus, or in the body of the stomach, on or near the greater curvature, are the most favorable for total extirpation, while in those at or near the cardia, radical removal is impossible. In cancer of the lesser curvature, not only are the symptoms for a longer time masked, but here, too, there is a greater tendency to secondary glandular involvement, thus decreasing the chances of radical removal.

The duration of unoperated cases varies from three months to two and one-half years, the average being about one year. On the other hand, Weil, of the Breslau Clinic reports non-recurrence for as long as twenty-one years after operation upon histologically confirmed gastric cancers, the Mayos, fifteen years, while the author has observed and reported 2 cases that have remained well five years, and a third one almost eleven years to date after radical operation.

Pathology.—The limitation of space precludes a detailed discussion of the pathological features of cancer of the stomach, which are of special interest only to the pathologist. For details of a pathological nature, the reader is referred to any standard work on pathology. The most common varieties of gastric cancer are the adenocarcinoma, the gelatinous or colloid, the medullary, the scirrhous and the sclerosing pyloric and diffuse fibrocarcinoma. Of these adenocarcinoma is the most frequent variety. It may form a solitary or a multiple tumor mass. It begins as a flat thickening of the mucosa which later may assume a polypoid form, though more frequently it ulcerates.

Gelatinous carcinoma favors the pylorus. It simulates adenocarcinoma, but is characterized by the production of a serous mucus which distends the alveoli, breaks them and infiltrates the connective tissues.

Scirrhus carcinoma in its severest form is the most extensive and metatastic type of gastric cancer, often attacking the entire stomach. It favors the lymph nodes and evidences itself by a diffuse thickening of all the coats and a cicatricial contraction of the stomach; it has a lesser ulcerative tendency than the other varieties. The hardness of this type of growth results from the large amount of connective tissue and relatively few cells.

Medullary carcinoma presents soft, spongy, greyish or yellowish white, fungoid masses that project above the mucous membrane. This form is very rich in blood-vessels and tissue cells and relatively poor in connective tissue. Hence it bleeds and ulcerates readily; moreover it has a tendency to early metastasis.

Sclerosing pyloric and diffuse fibrocarcinoma gives the so-called leather-bottle stomach, linitis plastica. The submucosa is chiefly affected.

SARCOMA OF STOMACH

Definition.—Gastric sarcoma is a primary or a secondary tumor which develops from connective tissue, smooth muscle fibres, lymphoid nodules or endothelial cells and subsequently invades and destroys other parts of the stomach. Unlike carcinoma, it is a non-epithelial neoplasm.

Etiology.—Nothing is known regarding the cause of sarcoma. Speculation here is even more fruitless than in the case of carcinoma, as sarcoma does not involve the mucous membranes in the beginning, so that trauma or irritation are excluded as possible causative factors. Though essentially a disease of children and young adults, available statistics indicate that primary sarcoma seldom attacks the stomach before the fourth decade of life and is most frequently found in that organ between the ages of fifty and sixty. The youngest recorded case was in a child three and one-half years of age and the oldest in a man of ninety-one. It appears with equal frequency in both sexes. Sarcoma forms five to eight per cent. of all primary malignant tumors, and from reports it appears that one-fourth of one per cent. of all sarcomas occur in the stomach. It must be remembered, however, that many cases diagnosed as carcinomas undoubtedly really were sarcomas; thus Perry and Shaw proved histologically that in a series of fifty cases classified in Guy's Hospital as cancers, four really were sarcomas.

Symptomatology and Diagnosis.—Active gastric symptoms may be lacking throughout the entire course, or at least until the process is far advanced. This arises from the fact that the mucosa is not involved and that there are usually no obstructive changes. In other cases the predominant signs and symptoms are those due to complications or to secondary changes in the growth itself, and these have given rise to most of the errors in diagnosis. When gastric symptoms appear, they vary from the mildest expression of dyspepsia to the severest manifestations of gastric cancer. No definite diagnostic criteria can be set forth. A tumor may or may not be present. Such a tumor is indistinguishable from cancer (q. v.). Correct diagnoses have almost always been made only after direct exploration. In a few instances a diagnosis was ventured after examination of tumor particles obtained from stomach contents, or after excision of a metastatic growth in the rectum, skin, glands, etc. Even the latter may, however, be misleading because of the possible intercurrence of cancer elsewhere in the body. In other words, even the demonstration of either sarcoma or carcinoma in another organ is no definite indication that the stomach condition is in correspondence therewith.

The first question which arises when a malignant tumor has been found is whether we are dealing with cancer or sarcoma. Both neoplasms have many characteristics in common, such as the ear-marks of malignancy (see section on Gastric Cancer), the alterations in gastric structure and functions, the occurrence of hemorrhages, metastases, splenic tumors and febrile states. Since cancer, however, arises from the epithelium, the mucous membrane is always affected, while in sarcoma it is usually not involved or, if so, only secondarily through pressure or erosion. Symptoms of secretory disturbances are therefore more typical of cancer and are not among the diagnostic criteria of sarcoma. Cancer is more frequently located at the orifices and in the body of the stomach; sarcoma more often along the curvatures; hence cancer is more apt to be associated with obstructive manifestations (stenosis). Matthes states that in diffuse lymphosarcoma on the other hand, the absence of stenosis or the presence of distention is a useful diagnostic indication, more particularly when the tumor is plainly palpable as a large growth. According to Haudeck, sarcoma does not develop into the interior of the stomach and makes no filling defect; in other words, the stomach presents a rigid wide lumen with constant dilatation. Where pyloric obstruction is present in sarcoma, it is usually a late complication and due to massive infiltration of the stomach walls rather than to a real pyloric stenosis.

Complications.—Sarcoma spreads chiefly through the blood vessels. The local complications may be perforations (in six to eleven per cent. of the cases, depending upon the seat of the sarcoma) and bleeding from large vessels (relatively rare). Metastases into remote organs are late and are more frequent with the round-celled type, and least frequent with the endotheliomas.

Treatment.—**Surgery** is the only therapeutic resource. It should be attempteed even where the diagnosis is doubtful. Resection should be made far into the healthy tissues. As in cancer, deep **x-ray therapy** may be tried postoperatively to prevent recurrence and also in inoperable cases. This procedure is still too recent for proper evaluation.

Prognosis.—The round-celled are the most malignant and the spindle-celled the least malignant types. The average duration of untreated round-celled sarcoma is reported to be about fifteen months; that of spindle-celled twenty-four to thirty-two months. The earlier the removal the less the chance of recurrence. Even the most prolific varieties, lympho- and fibro-sarcomas, are less apt to recur after operation than in the case of cancer.

Pathology.—According to their tissue origin, sarcomas form respectively true fibrosarcomas, leiomyosarcomas (malignant leiomyoblastomas), lymphosarcomas (malignant lymphoblastomas, Hodgkin's disease), and endotheliomas. According to their cellular structure, they are classified as small and large round-celled and small and large spindle-celled sarcomas. They may occur as nodular or diffuse hard flat tumor masses within the wall of the stomach, or as polypoid-shaped projections from the wall of the stomach into its lumen or into the greater or lesser peritoneal cavity. Their size may vary from a minute nodule to an enormous mass weighing, as in Baldy's case, fifteen pounds and almost filling the peritoneal cavity.

BENIGN TUMORS OF THE STOMACH

Definition.—Gastric benign tumors are circumscribed enlargements of normal glandular or cellular tissue elements. They may originate in any of the layers of the stomach and remain restricted to such layers or spread therefrom. Their growth is slow and they have no tendency to form metastases or to recur after removal.

Etiology.—The cause or causes of these tumors are still undetermined. The occurrence of mucous polyps has been ascribed to chronic alcoholism, chronic gastritis, atheroma of the gastric vessels and other chronic nutritional disturbances of the mucosa, but proof is still lacking that any one of these is a definite cause.

FREQUENCY.—Primary benign growths of the stomach are relatively infrequent. Eusterman and Senty, writing from the Mayo clinic, state that benign tumors constituted only 1.3 per cent. of all the 2,168 gastric tumors that came to operation between the years 1907 and 1921. This does not, however, indicate the actual ratio between benign and malignant tumors, as many cases of benign tumors undoubtedly were misdiagnosed as malignant ones and therefore regarded as inoperable. Ebstein found mucous polypi in the stomach at fourteen out of 600 autopsies, and this affords a clue as to how often the condition probably remains unrecognized.

Symptomatology and Diagnosis.—Depending upon the site, size and nature of the growth, the clinical picture ranges from euphoria with complete absence of gastric symptoms to an extreme state of suffering closely simulating cancer, for which disease these benign growths are so generally mistaken. Intramural growths not near the pylorus may be almost symptomless, though the very large ones—usually palpable through the abdominal parieties—may show themselves through a sense of epigastric weight and dragging with dyspeptic manifestations, loss of appetite and weight, anemia and even pain. With a growth situated in the pyloric region, especially if pedunculated, active dyspeptic symptoms are more frequent. Growths of this kind tend to prolapse into this opening and cause partial or complete obstruction with corresponding physical disturbances—gastric distention, food retention, gastrosuccorrhea and peristaltic rigidity. Frequently the vomitus contains blood. The obstruction, usually the first active manifestation of the disease, occurs through a ball valve action and in most cases is only temporary, the tumor falling back into the stomach and a period of quiescense ensuing, followed by new attacks of acute obstruction until death or operative interference occurs. Hemorrhage is a frequent symptom of tumor involving the mucous membrane, and is due to excessive vascularity of the growth or the surrounding mucosa, or to vascular erosion through ulceration or sloughing. Blood may be visible or occult in the stomach contents or stools. A relatively large number of benign tumors with ulceration present a clinical picture of peptic ulcer, for which condition they have frequently been diagnosed. Gastric secretion and motility should be normal where the mucous membrane is not involved and where there is no plyoric obstruction; otherwise, there may be an achylia, associated particularly in mucous polyps with an excessive production of peculiar mucus, resembling egg-white, such as might be expected with great multiplicity of goblet cells. Such a finding in the stomach washings should, as pointed out by Myer, arouse a suspicion of polyposis. The one sure means of establishing the diagnosis is, of course, the accidental finding of a tumor fragment in the vomitus, stomach washing or feces.

Aside from a lack of familiarity on the part of the profession with these conditions, the reason why these cases are so rarely correctly diagnosed and remain unrecognized, or afford operative or postmortem surprises, is that a shadow defect revealed by the x-ray is promptly assumed to be that of an ulcer or cancer. The first publication on the radiological diagnosis of benign tumor of the stomach, without prior finding of tumor fragments in the stomach washing, was that of the writer in 1916. Careful search of the literature fails to show a prior report of any case in which the diagnosis of benign tumor was made before operation purely on the roentgenological evidence alone. Since then, Holmes, Merrill, Carman, and Bryan have reported benign tumors preoperatively diagnosed in this way.

The Mayo Clinic report, cited above, describes 27 cases only 2 of which the benignancy of the tumor appears to have been radiologically recognized prior to operation. This is all the more surprising because some of the plates (Figs. 3, 5 and 7), which are reproduced with the paper quite strongly indicate a benign tumor. (Fig. 195 in Carman's book, though typical of a benign tumor was diagnosed and operated on for carcinoma of the stomach in 1919.) Surely the subject is of sufficient importance to engage the more earnest attention of the profession that we may not continue to condemn as having inoperable cancers, those patients who would be saved by a timely exploratory gastrostomy and the removal of a benign tumor. While experience is hydatid, blood, lymphangiomatous or chylous cysts, and cysts formed through the degeneration of new growths.

TUBERCULOSIS OF THE STOMACH

Definition.—An ulcerative or inflamatory granulomatous process due to primary or secondary tubercle bacillus infection of the stomach.

Etiology.—Gastric tuberculosis may be primary or secondary. The great majority of cases are secondary. In 1913, Zesas reported on primary cases from the literature, a large number of which are beyond question. Why does tubeculosis infection occur so rarely in advanced pulmonary patients who are constantly swallowing sputum containing enormous quantities of tubercle bacilli, while ordinary peptic ulcers due to streptococci are so common? As is shown by the work of Frank, Strauss, Wuerz and Fischer, the acid gastric juices afford little, if any, protection against the tubercle bacillus, which is acid-fast. The probable explanation is that the streptococcus is a rapidy growing organism, so that stomach motility does not prevent its settlement, while the extremely slow-growing tubercle bacillus cannot obtain lodgment and form colonies under such unfavorable conditions, if the stomach lining is intact. That intestinal tuberculosis is so much more frequent is undoubtedly due to the fact that the tubercle bacilli can remain in the intestine a far longer period of time, particularly in wall-adherent particles of feces, and can gain entrance at inflamed or eroded spots. Eisenhardt, in 1,000 autopsies found 566 secondary intestinal tuberculosis, and only one of gastric tuberculosis. Glaubitt, in 2,237 autopsies on tubercular individuals, found gastric tubercular conditions 47 times; Simmonds in 2,000 similar autopsies, only eight times and Gossmann 18 cases in 2,360 autopsies.

Secondary gastric tuberculosis may also occur by transmission through the lymph or blood channels, particularly in cases where the lesion is on the peritoneal surface. Arloing, on the basis of his experimental investigations, regards this as the commonest mode of infection. He found that every time he injected tubercle bacilli into the blood stream, he produced ulceration of the gastric mucous membrane, while the introduction per os did not cause an infection.* Apparently, gastric tuberculosis occurs slightly more often in males and favors the ages between fourteen and twenty-eight years. The belief long held that cancer confers immunity against tuberculosis is no longer tenable in view of numerous observances of the coexistence of both diseases in the same individual. A case illustrating this is published by Simmonds; here cancer had existed before the tubercular process was observed. Lyle reported another interesting case in 1913 with a summary of reported cases of combined tuberculosis and cancer of the stomach.

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* Unfortunately Arloing's original publication is not accessible to the writer. The above citation is from Spengler and Zesas who fail to state whether the experimental ulcers were proven tubercular or not, a most important point.

Exogenous Causes.—Infected food, meat, milk and butter are assigned by some as definite sources. Zesas carefully reviews this question with detailed citations of published laboratory investigations and case reports. Tubercle bacilli have been repeatedly found on the lining of normal stomachs, and must have reached there through the mouth. Eisenhardt found that the process of pickling does not kill the tubercle bacillus.

Symptomatology and Diagnosis.—There are no pathognomonic symptoms of the gastric tuberculosis. The disease is seldom correctly diagnosed before operation or autopsy. The ulcer types closely simulate those of ordinary peptic ulcer, although they are apt to be much more painful, and appear to have a lesser tendency to perforation, bleeding or healing. Tuberculous tumors have usually been mistaken for cancer, since both types of diseases have many manifestations in common. In both the clinical history is short and marked by emaciation, vomiting, lack of appetite, progressive weakness, and increased dyspepsia after solid food. The gastric contents in tuberculous tumor very frequently show absence of hydrochloric acid and the presence of lactic acid, as well as of occult blood, coffee-ground sediment and stagnation of contents. The x-rays, too, may show shadow-defects like those of cancer. A diagnostic point is that the period of predilection for cancer is after the age of forty, while that for gastric tuberculosis is before thirty. While a positive diagnosis of gastric tuberculosis can only be made from direct, gross or histological inspection, the clinical picture of the tumor variety of the disease is one demanding surgical exploration. If symptoms of gastric ulcer, pyloric stenosis or cancer are present in a patient with tuberculosis of some other part of the body, the probability that the gastric lesion is tuberculous, rather than otherwise, should always be borne in mind.

Complications.—These have in large part already been discussed. They include hemorrhage from erosion of gastric vessels, perforation, partial or complete, (the former is frequent, but the latter rare, because of reactive peritoneal thickening) perigastric adhesions, which may be slight or be extensive enough to form tumor masses and even to cause partial or complete stensis of the stomach, especially at the pylorus; gastro-duodenal fistula from rupture of a caseated focus (Mathieu and Remond) and lymph gland enlargement. This last is almost always present with a tubercular process. It may be slight but often is very extensive. When involving the pyloric or duodenal regions, it can also cause obstruction of the lumen.

Treatment.—This should be directed not alone to the gastric lesion, but also the underlying specific causative factor must be recognized. In addition to the customary medicinal or surgical treatment for the relief of the gastritis, ulcer, obstruction, tumor mass, etc., consideration and employment of available general and special therapeutic measures to combat the tuberculosis must be restored to. These last are described in fullest detail in Vol. II of this work. Attention is directed especially to the rules and precautions to be followed in the use of tuberculin for therapeutic and diagnostic purposes.

Internal measures should be given first trial in the gastritis and uncomplicated ulcer cases, following along the routine lines of lavage, diet, rest, drugs, etc., as discussed elsewhere in the chapters devoted to those conditions. Advanced cases, as well as those in which surgical procedures are too hazardous, must be left to the internist for such relief as can be given. Surgery is especially indicated for obstructions, tumor masses, lymphadenitis, adhesive peritonitis and for ulcerous conditions that have resisted internal therapy or that are associated with recognized surgical complications.

Prognosis.—Unfortunately, sufficient data are at present not available upon which to base a correct opinion as to the real value of local and general internal measures, even in milder types of gastric tuberculosis. On the other hand, enough cases have been reported to fully justify the conclusion that timely and proper surgical intervention has proven of the greatest benefit, and, in those desperate conditions so closely simulating cancer, even life saving.

Since, however, the great majority of cases are suffering also from tuberculosis elsewhere, especially in the chest and intestines, we must recognize that except in the strictly primary types the cure of the local gastric lesion is by no means a cure of our patient. Due consideration and treatment must also be given to these tubercular conditions in other parts of the body. Upon their existence and amenability to relief or cure will depend the ultimate outcome of the case. As long as they persist they remain a menace to reinfection of the stomach.

Pathology.—Gastric tuberculosis manifests itself anatomically as ulcer, miliary tubercle, tumor-like masses and perigastric processes. In all types, the pyloric region is the seat of predilection. The explanations advanced for this are the relatively greater activity of this part, its susceptibility to traumatism, and hence to even slight degree of infection. Virchow, Rokitansky, Klebs and other adherents of the lymhogenous theory of infection are of the opinion that the greater supply of lymph follicles in this region is the cause. The greater curvature is the part next most frequently affected.

(1) *Ulcers.*—These are generally single, occasionally multiple. They are mostly round or slightly oval, their borders being irregular, fissured and undermined, and occasionally they have miliary nodules or papillary protrusions at their periphery. Miliary tubercles also occur in the serous coat opposite the ulcer. The ulcers usually extend only to the submucosa and seldom involve the muscular layer. Their base is grayish-red or pale and granular, often caseous. Microscopically, giant cells, tubercle bacilli and, at times, necrotic foci have been found in the periphery. Endarteritic and endophlebitic conditions may prevail. As a rule the ulcers are small sized, but to this there are striking exceptions. Thus, Musser reported a tubercular ulcer about three inches long and one inch wide, and Baetzner one the size of the palm of the hand. Tubercular ulcers rarely form scars, but Curschmann, Trilier, and Labadie-

Lagrave found scars of this type at autopsy; while Bardasch mentions 4 cases where new ulcers and scars of ulcers coexisted.

(2) *Miliary Tubercles.*—These are rare. They appear either as the solitary tubercle found on the inner wall of the organ and apparently a direct contact result from swallowed tubercle infected sputum, food, etc., or else in the form of the well-known multiple miliary tubercle scattered throughout the organ and assumed by many writers to be a secondary infection through the blood stream.

(3) *Tumor-like Masses.*—These result from inflammatory hypertrophic and hyperplastic changes to which are added lymphomata, perigastric adhesions to adjacent parts and organs and other similar secondary pathological involvements. The tumor masses vary greatly in size and location and are prone to produce stenoses of the pylorus. Quite frequently, too, they have been operated on under the mistaken diagnosis of cancer.

Pancet and Leriche described an inflammatory form which they called "tuberculose inflammatoire." This may affect either the epithelium or the submucosa. If the former, a more or less widespread glandular (adenomatous) proliferation may arise. Involvement of the submucosa is productive of a diffuse, hard inflammatory infiltration or of a circumscribed tumor mass with stenosis.

The hypertrophic type of "tuberculoma" of the stomach is rare. It is said to favor the younger ages. It is not possible to give a distinct pathological picture of the condition. According to Spengler it forms an infiltrating annular swelling at the pylorus, which frequently at first ulcerates superficially and later progresses to form a real ulcer at right angles to the pyloric axis. Nodules are at times found in the ulcer base.

The hyperplastic form involves the deeper layers of the gastric wall, which becomes considerably thickened. On section the cut surface of the stomach wall is perfectly smooth, firm and dead white. Microscopically the diseased tissue shows superficial erosion of the epithelial tissues, extensive round cell infiltration (lymphocytes and plasma cells), giant cells and at times tubercle bacilli. There is, too, extensive lymph gland enlargement along the greater and lesser curvatures of the stomach.

(4) *Perigastric Processes.*—These have already been mentioned. The adhesions may so constrict the lumen of the stomach as to produce even a complete stenosis of the pylorus. With extensive adhesions there may be a matting together of the stomach and adjacent organs and parts into a large irregular and more or less dense and inseparable tumor mass.

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CHAPTER XVII

ATONY OF THE STOMACH, DILATATION OF THE STOMACH, PYLORIC STENOSIS, STENOSIS OF THE PYLORUS IN INFANTS, DUODENAL DILATATION, DUODENAL STENOSIS

BY C. F. MARTIN, M.D., AND R. H. M. HARDISTY, M.D.

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ATONY OF THE STOMACH

Synonyms.—Motor insufficiency; myasthenia gastrica; hypomotility; and gastrectasis.

Nomenclature.—Some confusion in the terminology of this condition will always exist unless one considers all of the etiological factors involved. Attention should be drawn to the fact that simple atony of the stomach is really not synonymous with gastrectasis or dilatation. It must be conceded that a so-called "primary atony" exists, be the condition temporary or permanent. As a matter of fact, atony of the stomach is always secondary to some cause, be it anatomical, physiological or pathological; e. g., a simple atony is extremely frequent where dietetic indiscretion follows general fatigue, a condition often commonly known as a "bilious attack." An identical state of affairs occurs often with migraine attacks. In such cases temporary fatigue results in some defect of the neuromuscular mechanism whereby the function of the gastric wall is impaired. Food is retained in the stomach until nature relieves the condition by emesis. Physiologically speaking, this is simple atony; there is motor insufficiency, i. e., hypomotility or myasthenia. The stomach is not necessarily intrinsically dilated, nor is there any obstruction at the pylorus.

A similar condition exists during convalescence from prolonged fever and in wasting diseases, the only difference being that in these latter some dilatation may occur through the longer duration of the etiological factors. The ultimate result is merely accentuation of the simpler process. Gastrectasis, i. e., a dilatation of the stomach following these conditions, is merely an exaggeration of the original atony, and as such is also to be classified as simple or primary gastrectasis in contradistinction to those cases of *secondary* gastrectasis where dilatation is due to pyloric obstruction.

General Considerations.—In this connection it is important to recognize the fact that the human stomach has no defined mathematical dimension; that normally one may have a very large viscus, much larger than what is accepted to be the normal, and yet the motor and secretory functions may be unimpaired. This condition is usually called "megalogastria."

Size and motor function are not necessarily correlated or inter-On the other hand, in the thickset, robust man, the viscus lies high, has a very definite relation to his build and his constitution, both as regards its shape and size, as well as its position, and even its motor power. Thus, the long, lanky person with a general myasthenic typus, will often present a low-lying, large and flabby organ, i. e., a potentially atonic stomach. On the other hand, in the thickest, robust man, the viscus lies high, has a good tone and is of smaller size. In the former case, the myasthenic individual, if he be neurasthenic, is certain to have gastric atony on the slightest provocation and is a likely subject for gastric neurosis. The question is one of constitution or type, to which, as a rule, far too little attention is paid.

Rossi and Zanon have drawn attention to the relation between gastric function and splanchnic type, i. e., the ratio of the size of the abdomen to that of the thorax on the one hand and the ratio of the length of the trunk to that of the extremities on the other. The so-called "microsplanchnic" individuals are those who are more liable to have a feeble muscular tone, and, therefore, a deficient motility. Vagotonic individuals, on the other hand, are those in whom there is excessive tonic innervation or where the sympathetic innervation is diminished.

Tonicity.—It must be remembered that, physiologically speaking, the tone of the stomach responds to pressure or distention, and, therefore, the presence of food is a sufficient stimulus to peristalsis. Tonicity is a necessity for rhythmical movement, and one may readily see by the fluorescence screen how tonic contraction and rhythmical peristalsis disappear where general myasthenia occurs. In these cases, so often the central nervous system has failed to stimulate tone. Cannon's work has revealed the effects of psychic conditions upon tone. This in itself is a very important factor in the explanation of the symptoms of gastric neurosis, and motor power is of far more importance in this respect than is secretion.

Form and position of the stomach depend upon its tonus to a very large extent, and this in turn depends on the vegetative nerve supply.

The gastric secretion, again, is largely dependent upon vagus innervation apart from hormones.

Physiology.—The stomach is an automatic organ, the extrinsic nerves being mere regulators of its movements.

The pyloric sphincter, with its circular fold of mucous membrane, shuts off the stomach from the duodenum. During the hours that solid food lies in the stomach, some of the thinner particles are expelled at intervals, the duodenum being shut off at other times. In other words, the pyloric sphincter relaxes periodically. The function of the pyloric end of the stomach seems to be to churn, titrate and expel food. Relaxation of the sphincter is favored by the presence of hydrochloric acid in the stomach. On the other hand, where hydrochloric acid reaches the duodenum, it favors the contraction of the sphincter. The presence then, of acid in the duodenum causes closure of the pylorus until the juice there is neutralized.

The fundus is the reservoir of the stomach, and the food may lie there denum, it favors the contraction of the sphincter. The presence, then, of stomach is kept in a state of tone during gastric digestion. It is well to remember that in the normal stomach there is never an empty space; in other words, the stomach closes upon its contents and upon the air in its upper portion.

With reference to dietetic treatment, it should be remembered that in the healthy stomach, carbohydrate food passes into the intestines much more quickly than do proteins. On the other hand, fats, when alone, remain longer in the stomach than any other form of food, and when mixed with other foods, they cause delay in expulsion. In the presence of subacidity, carbohydrate food leaves the stomach more quickly, while the albumin and fatty foods are less effective.

Absorption in the stomach is slower than it is in the intestines. Among the substances that are absorbed in the stomach in a greater or less degree are water (very slightly), salt, sugars, dextrine, proteoses, peptones, and some soluble or liquid substances like drugs, alcohol, etc. This is of importance both for diagnosis and therapy.

Etiology of Simple Atony.—Simple atony, as its name implies, is a form of motor insufficiency where the muscle is lacking in tone. Emptying time is delayed; there is no dilatation, or else the dilatation is temporary and does not exist when the stomach is empty.

In atonic gastrectasis the muscle is abnormally stretched by the burden of its contents. The normal elasticity and tone are lost because of the functional weakness of the neuromuscular apparatus. One of two things happens—either the motor nerves are less easily stimulated or else the muscle reacts more slowly and tires more quickly. In other words, the muscle contracts more slowly and with less intensity around the contents, so that expulsion is delayed. This condition occurs in individuals who are weakly from any cause, from continued over-burdening of the stomach and from nervous depression. All psychopathic states are apt to act upon unstriped muscle, as has been emphasized by Pavlow and Cannon.

The etiological factors, then, in the production of simple gastric atony may be enumerated as follows:

Firstly, nervous conditions; constitutional diseases; general malnutrition and anemia; chronic diseases of any kind; acute diseases and infections, and the convalescent period.

A second group of cases are the result of dietetic indiscretions as regards quantity and quality of food, and excessive use of alcohol in any form. Defective teeth and insufficient mastication may well come under the category of dietetic factors.

A third group of cases include those where persistent fatigue, late hours, insufficient rest, or vice versa, insufficient exercise, and irregular habits with reference to the toilet, militate against the normal functioning of the neuromuscular apparatus.

Malposition of the viscera is another cause, so that gastroptosis and enteroptosis are frequently found associated with a mild degree of atony, and in this group to a certain extent may be included the results of pregnancy. Aerophagia, or the habit of swallowing air, is another common cause.

Diagnosis.—TESTS OF MOTOR POWER.—For the most part tests of this function depend on evidence of delay in the expulsion of contents.

PRECAUTIONS CONCERNING THE TESTS, THEIR SIGNIFICANCE AND INTERPRETATION.—Many factors must be taken into consideration in testing for motor power. In the first place, the significance of a delay in propulsion of food is often hard to determine and one must not overestimate the importance of a slight impairment of motor function. The following points must be kept in mind:

(1) There are all degrees and transitions of motor insufficiency.

(2) The mere removal of food from the stomach is not necessarily the equivalent of its ejection by the sphincter pylori (Wheeler).

(3) The rapidity of emptying may temporarily alter the tonicity of the stomach, as well as the acid concentration of any one sample.

(4) The duodenal regurgitation may be present, and may deceive one as to the amount of residue in the stomach after the given meal.

(5) The point of the tube used for examination may be shifted by the motility of the stomach, and in this way faulty conclusions may result.

(6) Different foodstuffs often affect the tone of the stomach differently, and the same may be said of the secretions.

(7) Sometimes the delay is purely ephemeral.

GENERAL EVIDENCES OF ATONY.—According to the degree of atony, so will there be more or less constitutional or local evidences of its presence. The tissues will be dry, there will be corresponding thirst and constipation, as well as oliguria. With reference to oliguria, three degrees of atony have been described according to the amount of urine that is passed. In the first degree of atony, 1000-1500 c.c. of urine are passed; in the second degree, 500-900 c.c.; and in the third (advanced type of atony) less than 500 c.c. urine are excreted.

Among the chief factors in modern gastric analysis that are used to determine motor power, the stomach tube and the x-rays are the most important. Other tests, chemical and physiological, are for the part of minor importance and obsolete. Reference to this will be made later.

THE USE OF THE STOMACH TUBE.—(1) *Fasting Contents.*—Normally the stomach is never empty in the early morning; there is a fair amount of fluid present—from 5 to 30 c.c. according to Martius, from 10 to 100 c.c. according to Schreiber. The fluid contains hydrochloric acid in a very small amount only, as most of it disappears in twenty-four hours. The presence, therefore, of acid in the fasting contents is significant of faulty evacuation. Reichmann's disease is to be considered as a prolonged secretion rather than a continuous hypersecretion. The fluid present in fasting contents may partly arise from the duodenum, the regurgitation being brought on through the stimulus of the tube. The chief feature of importance in the pathological state of any fasting contents is the presence of *recognizable food particles.* (It is important to make repeated examinations to see that this condition is persistent.) Whenever food particles are persistently found in the fasting contents in the early morning, there is only one conclusion to be made; *viz.,* that the motor power is defective and evacuation from the stomach is not carried on normally.

(2) *Technique.*—Examination of the fasting contents may be made by means of the Rehfuss tube, or similar tube such as is commonly used for fractional analysis. Those tubes with very small metallic tips are much more easily swallowed than are the larger tubes, and sufficient contents may be removed by a withdrawal syringe to enable one to make whatever tests are necessary.

(3) *Examination after Test Meals.*—Various meals are employed to determine the motor power of the stomach. The simplest meal known is the test breakfast, consisting of a slice of dry toast with a cup of water (10 oz.)*. Shredded wheat may be used instead of the toast. The stomach should be empty in two hours. At the end of one hour, there should not be more than 100 c.c. present, and at the end of an hour and a half, 40 c.c. should be the amount of residue.

The test dinner, consisting of soup, meat (160 gm.), mashed potatoes (50 grams), a glass of water, and a roll, is also used to determine the motor power. The stomach should be empty in seven hours. A few raisins may be added to this meal. This meal may be given in the evening and the stomach examined the next morning to test for the possible retention of food. Three degrees of motor insufficiency are established by these tests:

(*a*) Mild motor insufficiency, often temporary. Test breakfast or test dinner shows some delay in evacuation. This condition is generally designated as "stagnation."

(*b*) Some food remains twelve hours after the test dinner, while some has passed on. This is called "partial retention," and is characteristic of early ulcer, cancer or obstruction, as well as of severe atony from other causes.

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*Normally the speed of evacuation is always greater than the speed of secretion, *i. e.,* there is never more liquid at the end of half an hour than the amount ingested. There is gradual diminution after half an hour until in two hours the stomach is empty.

(*c*) Permanent retenton, i. e., little or no food leaves the stomach. This condition is associated only with pyloric obstruction.

X-Ray Methods.—The many and futile discussions as to the merits and demerits of x-rays in the diagnosis of gastro-intestinal diseases would, perhaps, be minimized if one could realize that certain essentials are required of every such investigation. The following points are emphasized:

(1) There must be a complete clinical history of the case.

(2) A fluoroscopic examination is an essential part of the x-ray diagnosis. This investigation must last for a period of at least fifteen minutes, and if necessary, be repeated at intervals.

(3) There is normally a wide variation in the evacuation time of the stomach. Plates taken during the digestive period give but a partial representation of the true state of the viscus.

(4) A skilled technician is essential, as is also a good clinician to coöperate with him. The pictures must be studied in series, and if necessary, a second examination in series should be prepared.

X-ray examination for atony of the stomach is no doubt better than the tube, though one must remember that a six-hour delay does not mean obstruction. There are many other causes of atony which would appear under examination by these means.

The standard meal adopted for x-ray examination consists of barium sulphate, 3 oz.; and a cereal (gruel) or milk, from 12 to 16 oz. The emptying time varies between three and six hours, the variations depending upon the type of individual and the state of the pylorus. What is slow in evacuation for one individual is quick for another, and this depends largely on the type. In the tall, thin individual, the tone is less than in the short and powerful person. Moreover, one must remember, as has been emphasized by Franklin White, that one factor may neutralize another, *e. g.*, rapid emptying time of achylia may be slowed by ptosis, or vice versa. Delay caused by an obstruction of the pylorus may be compensated in part by the powerful peristalsis of a strong person.

Fluoroscopic examination is the most important. Normally the stomach, when empty, is collapsed on its gas-bubble; the walls are apposed as the barium meal enters, the shadow flows through the bubble to the pars media, where it remains but a short time, and then gradually opens this portion. It then descends and gradually collects in a half-moon form, with its broad level of food, until both ends of the crescent are filled. The filling depends upon the tone of the wall. On the other hand, one must remember that these signs are sometimes not reliable, because with a relaxed abdominal wall, the barium often drops quickly, going sometimes straight to the bottom, where it collects. Thus, x-ray pictures of the so-called "atonic long stomach" show a long drawn-out organ, the bottom reaching into the pelvis; the organ is larger and narrower in the middle, the bismuth remains at the bottom, the walls above are more or less closely pressed together, and the air-pocket, instead of being rounded, is more pear-shaped. This differentiates atony from mediogastric spasm, in which the bismuth is in two portions, one under the air-pocket and one at the bottom. Peristalsis at the bottom is absent, or deficient and delayed. It is sometimes difficult to differentiate this from mild stenosis or prolonged pyloric spasm, and one must be aided by physical findings.

X-RAY PICTURE IN MARKED ATONY.—There is no resistance whatsoever to the entrance of the bismuth; it falls as though into an empty bag into the greater curvature. Here it remains, not filling up the stomach as a whole; sometimes a very small quantity is left in the fundus and in the rugæ. Peristalsis is very superficial. The position of the pylorus is about normal. In gourmands, the capacity of the stomach may be increased without delay in the gastric motility.

Treatment.—PROPHYLAXIS.—Avoidance of all the etiological factors is the main consideration. Susceptible individuals, *i. e.*, weaklings, neurotics, those of the splanchnic type, etc., should be **careful of their diet,** both as to quantity and quality, as well as to the manner of eating. The same applies to convalescents from fevers and those afflicted with chronic diseases, where the musculature of the stomach is enfeebled and more easily dilated than is the normal. The **teeth** should be carefully attended to and any tendency to **constipation** should be relieved.

DIETETIC TREATMENT.—This is by far the most important of all, inasmuch as the atonic stomach does not empty readily and its expulsive power is less than normal. The **diet** should consist of small and highly nutritious meals served in the most easily digested form possible. The directions for diet depend largely upon the degree of atony present, as well as upon the type of dilatation. It is only in the very severe cases that the patient must remain in bed, though in the mild cases it is well for patients with atony to **lie down for half an hour** after taking food. In this way, the digestion is aided, and the food exercises no downward drag on the stomach, and passage into the intestines is assisted, all the more so if the patient lies in the right lateral position.

In ordinary cases of atony, sufficient food must be taken, and as the meals must be small, the feedings should be more frequent. **Five small meals a day** are better than three large ones. The kind of food depends to some extent upon the secretion. If, for example, there be hypersecretion or hyperacidity (which usually implies early obstruction or irritation) it is well to consider the need of a **protein diet** and the avoidance of excessive carbohydrates and sugars. The following diet list may be used in milder cases where hyperacidity exists:

SOUPS—In small quantity and clear. Take beef, mutton or chicken broth, or oyster soup, strained vegetable soups. Avoid rich soups and chowder, tomato soup.

FISH—Take boiled white fish of any kind; haddock, halibut, cod, etc., with cream sauce, or broiled (not fried); oysters. Avoid salmon, shrimps, sardines, crabs, lobsters and salt mackerel.

FARINACEOUS FOODS—All bread to be at least twenty-four hours old. White and brown bread, Graham bread, toast, crackers, cream of wheat, rolled oats, shredded wheat, hominy, milk toast, sago, tapioca, rice and arrow-root. Avoid fresh bread, hot rolls, corn bread and johnny cake.

FATS—Fresh butter, or washed in water; nothing fried.

EGGS—Boiled, poached, or raw. No fried eggs.

Meats—Once daily. Take beef (without gristle or fat, very little gravy), mutton, lamb (no fat), chicken, turkey, guinea hen, partridge, cold boiled ham in small amount. Avoid fried meats, veal, pork, liver, corned meats, goose, duck, sausages, kidney, hashes.

Vegetables—Take a little of baked or mashed potatoes, soft-boiled green vegetables, spinach, green peas, French beans, asparagus, artichokes, cauliflower, lettuce, cress. Avoid cucumbers, beets, parsnips, egg-plant, turnips, carrots, squash cabbage, sweet potatoes and brussels sprouts.

Beverages—Take milk, weak tea, cocoa, buttermilk, Vichy, Poland water, and a little light burgundy. Avoid ice water, malted liquors, sweet wines and spirits. Avoid sour, spiced or peppery foods, salt foods, pickles, sweets, fried foods, very hot or ice cold drinks.

In cases with lowered acidity, the quality of diet is less important than the quantity, the one proviso being that no food be given which tends to cause fermentation. For this reason, one should **limit the amount of carbohydrates** given, as well as of rich foods. Of all the carbohydrates, lactose (which may be added to the milk and other foods) is the most suitable form. Fluids should be restricted.

Drugs.—Certain drugs are useful in the treatment of these conditions of atony or dilatation in addition to the dietetic and hygienic measures. They may act locally or through their systemic effect.

In atony and dilatation without stenosis, **strychnin** in some form is very useful, either as tablets of strychnin sulphate, grain 1-30 (.00216 gram), taken half an hour before meals, or as tincture nux vomica, with which may be combined tincture gentian compound, and tincture of quinin. Other drugs used in the same way are the **bitter vegetable extracts**, as tincture condurango, a very useful preparation, and tinctures of cinnamon, cardamons and rhubarb. By these means we can often stimulate appetite and gastric peristalsis in neurotic and over-fatigued individuals or in convalescents after prolonged illness.

The use of hydrochloric acid in cases of diminished or absent acidity is not satisfactory. In order to have the amount of acid approximate what is normally in the stomach, we have to give very large doses and repeat them during the period of digestion, and this is difficult to carry out and is not satisfactory. When, however, we give it in small doses of from 10 or 15 minims (.6 to .92 c.c.) of the dilute acid in water before meals, it seems to stimulate appetite and secretion, and so can be used as the bitter vegetable extracts or as a stomachic.

A wineglassful of some **good dry wine** such as **sherry** or **madeira,** to which may or may not be added a few drops of **bitters,** is also often of value in stimulating appetite when taken a short time before meals. The ordinary cocktail, so highly thought of in social gatherings, has the disadvantage of being over-stimulating and irritating to the gastric mucosa.

The use of pepsin is not advocated. The various pepsin preparations are not as a rule satisfactory, so that if we wish to use pepsin, it is best given in from 3 to 10 grain (.195 to .65 gram) quantities of the original scale form dissolved in dilute hydrochloric acid. Pepsin, it must be remembered, is generally present in the gastric secretions even when hydrochloric acid is greatly diminished or absent, so that we need only supply the dilute hydrochloric acid in order to get the protein digestive action.

Prognosis.—Prognosis is largely determined by the degree of atony present, by the nature of the etiological factor, and the possibility of overcoming it; and lastly, the outcome is dependent upon the type of constitution possessed by the affected individual. The milder cases respond readily to treatment. In the severer cases, the duration is longer on account of the greater severity of *the causa defecto.* It may be said in general that where patients respond within a reasonable time to dietetic precautions and general hygienic measures, the outlook is much more favorable. The graver cases with general myasthenia do not do so well.

SEVERE DILATATION OF THE STOMACH AND PYLORIC STENOSIS

Types.—Dilatation of the stomach may be acute or chronic. (The acute cases are discussed in a subsequent section.)

Chronic Dilatation of the Stomach and Pyloric Stenosis.—In the early stages the clinical history is that of simple atony, but as the disease develops the picture changes, due to the fact that the dilatation increases, the motor power becomes more impaired and the signs are those of gastric retention.

One may classify all these cases under two general headings—those with and those without pyloric stenosis. It is not always easy to differentiate these two in the earliest stages; moreover, with better methods of examination and with improvements in the technique and understanding of x-ray methods, it has been found that the cases of chronic dilatation without stenosis are much less common than was previously supposed.

Chronic Dilatation Without Stenosis.—These cases are more frequently found in adults, though seen at times in infants affected with rickets.

ETIOLOGY.—The cases are for the most part but an exaggeration of those that occur in simple atony, as above described. In the present article the subjects of chronic dilatation and pyloric stenosis will be taken up jointly. Three groups occur:

(1) Simple atony, which occurs as a result of mild dietetic indiscretions or from temporary pyloric spasm.

(2) Severe degrees, no stenosis or merely one of a temporary kind. In this group are included the cases resulting from chronic gastritis, spasms of the sphincter pylori, defective secretion leading to delayed evacuation: the dilatation usually persists and is more than temporary.

(3) Stenosis of the pylorus, continuous or malignant.

SYMPTOMATOLOGY.—In mild cases, the symptoms may be absent, or there may be merely fulness, discomfort after meals, especially after large meals, flatulence, but no vomiting or pain; the appetite may be good and the general health unimpaired. In these cases the severity may depend upon the state of the indiscretion, and, as a rule, fluids or solids are of equal significance in the pathological factors. The symptoms all disappear after the evacuation of the stomach.

Severe Degrees.—The symptoms depend upon the etiology and the degree of atony present. Where no stenosis has existed, the factors are chiefly gastritis and spasm of the pylorus. The symptoms occur as in the milder cases, and to these others are added, nervous symptoms, headache, depression, dizziness due to so-called "auto-intoxication," tetany. Vomiting may occur, bulky in amount, sour and fermented, and at long intervals. There may be thirst, obstinate constipation, diminished urine, dry skin and mucous membrane, and emaciation.

Physical Signs of Dilatation.—By inspection sometimes the stomach may be outlined beneath the skin if the patient be not too stout and if the viscus be distended by gas or food. Visible peristalsis is present only with stenosis.

In palpation one can often feel the outline of the stomach in advanced cases. Fluctuation is felt, especially with stenosis. The succussion splash may occur according to the degree of dilatation. It must be remembered, however, that the splashing sound is only of relative importance; it is often present with mild atony when dilatation is absent, and it gives no idea whatever of the degree of dilatation. Needless to say, succussion splashing and clapotage are likewise unimportant until the tests are made at a time sufficiently remote from any previous ingestion of fluid. It is also less important for neurotics, with sensitive people or those with rigid abdominal walls.

Intermittent hardening of the stomach or "stomach stiffening," has reference to a sign emphasized chiefly by the French authors in cases of pyloric stenosis where visible peristalsis is present. Periodical rigidity occurs.

Percussion is often of dubious value for outlining the stomach. As a rule, it is of more use for the upper border than for the lower. Auscultatory percussion is valueless.

Insufflation is sometimes of assistance in outlining the stomach in thin people, but considering the unimportance of position and shape in gastric diseases, this method of examination is more or less obsolete.

Treatment and **prognosis** have already been discussed under atony of the stomach.

HISTORICAL.—Fabrico d'Aquapendente was among the first to show that dilatation of the stomach was possible. This, however, was an anatomical study on the cadaver. Duplay here apparently made the first clinical study of this condition from an autopsy in 1833, while Widmann, ten years later, was among the first to describe a pyloric stenosis from cancer and discuss the possibility of dilatation without stenosis. Cruveilhier's work in 1852 forms one of the masterpieces of clinical study with reference to hypertrophic and atonic conditions of the stomach. It was he also who laid stress upon the inspection and dilatation of the abdominal wall in diagnosis of these conditions. Kussmaul, in 1869, invented the stomach tube, while Penzoldt, in 1875, gave his monograph on "Dilatation of the Stomach," which brought up to date the collected experience of all previous authors and established the condition as a definite clinical entity.

Dilatation from Pyloric Stenosis.—ETIOLOGY.—The stenosis may be intrinsic or extrinsic.

The intrinsic factors may be parietal or in the canal. Cancer is the commonest of all and embraces all varieties of stenosis,—tumors, vegetations, infiltrations, linitis, ulcerocancers. The site of election for all these is the pylorus. Two-thirds of all known cicatricial stenoses are due to ulcer. The ulcer may be active or cicatrized; it may obstruct by itself or cause obstruction in the vicinity by traction or retraction, or because of perigastritis, or obstruction of distant parts. Other cases of pyloric stenosis include syphilis, tuberculosis, congenital pyloric stenosis, fibromata and polypi (Cornil. Chiari), foreign bodies (Bouveret), gall-stones (White).

Among the extrinsic causes is compression, *i. e.*, liver changes, with alteration in the anatomical position; cancer of the gall-bladder and ducts; cancer of the glands; tuberculous glands of the hilus, and pancreatic cancer. Adhesions are more common as a cause, e. g., local peritonitis, from local inflammation of the stomach, duodenum or gall-bladder. It is difficult to find autopsy cases where there are no adhesions in this region; sometimes they are like thick ligaments; they vary in number, extent, organization, and look as though they were inflammatory, as they probably are. Most of these adhesions, indeed, are relics of old perigastritis, duodenitis or cholecystitis. Sometimes the adhesions are so extensive as to cause total symphysis or synechia gastrica.

DIAGNOSIS OF PYLORIC STENOSIS.—According to Hayem's law, anyone having persistent residual fluid in the stomach in the early morning, with food particles, has pyloric stenosis.

Examination of the fasting stomach is of capital importance, especially for early diagnosis. In addition to this, there is the history, characteristic vomiting, inspection, palpation, dilatation, intermittent stiffening, perhaps peristaltic waves, and tumor.

In mild cases, diagnosis is less easy; it hangs upon the interpretations of increased fluid in the fasting stomach without the presence of food. In Reichmann's disease, the so-called "disease of supersecretion," the fluid is persistent, very acid, there may be little or no food present, and the findings are recurrent after a lavage on the previous evening. The modern view of Reichmann's supersecretion tends to record it as prolonged secretion rather than a continuous one, and that it is more often associated with ulcer than has been realised. One thing is certain—that pure hypersecretion is rare in the presence of ulcer.

ETIOLOGICAL DIAGNOSIS OF PYLORIC STENOSIS.—Pyloric stenosis from malignant disease will show a residual fluid with little or no free hydrochloric acid. The gastric juice after meals is devoid of pepsin; the acid present is due to higher fatty acids, and is often much increased, due to the presence of lactic and butyric acids. The residual fluid is often brownish, colored from the presence of old blood and may resemble coffee grounds; it is often foul in odor. There is history of recent illness, or a recent exacerbation; there is anæmia, the loss of weight, a pain and other local symptoms, and the x-rays may reveal through the barium meal a condensation of some part of the gastric musculature, and perhaps a filling defect.

Benign pyloric stenosis, as a rule, shows retention of fluid; is hyperacid; there is little or no fatty acid; the odor is bland, even where much food is present; pepsin and hydrochloric acid are both increased; sarcinæ and yeast cells are often found; the history is that of an old affair, with painful crises, vomiting, etc. The signs are those of ulcer.

Where an old ulcer shows transition into malignancy, there may be a change from a hyperacid state into one of progressive hypo-acidity, and ultimately the presence of lactic acid. Boas-Oppler bacilli may be present.

THE SYMPTOMS OF STENOSIS ITSELF.—Pain is always present, except in very mild cases. It varies in intensity and may be very severe; it is continued or intermittent and may be periodical, resembling gastric crises. Vomiting is irregular at first, related to meals, and ceases if meals are regulated, or at all events, is less common. Later, with the increasing stenosis, the vomiting becomes habitual, until at last it is regular every 24-48 hours. Still later, with gastrectasis, intervals are longer, and as the atony increases, the stomach cannot completely empty itself and vomits only the excess of fluid. Rarely the stomach remains intolerant and vomiting is frequent, and dilatation does not occur.

The amount of vomitus varies. The more frequent the vomiting, the less the amount of fluid. In severe cases, there may be an excess of 3 or 4 litres (6.3 or 8.5 pints). As a rule, vomitus is semi-liquid, with copious food remnants from meals that have been taken several days before. Indeed, food particles may remain for weeks before they are rejected. Fermentation is copious; there is an odor of butyric or acetic acid, and putrefaction sometimes occurs. Hence the greater acid cöefficient which is often present.

Intermittent Hardening and Peristaltic Waves.—The intermittent tension of the epigastrium, as described by Bouveret, is not always present, but occurs usually if the stenosis is pronounced. It is rare with ptosis alone. It is possibly associated with some kinking of the gastroduodenal angle; it occurs usually after meals and is sometimes prominent where pronounced stenosis and much dilatation with tolerance are found.

If one empties the stomach with a tube, the symptoms disappear, because one needs intra-gastric tension for their production. The symptoms may be induced by mere exposure of the abdomen to the air, or by the application of a wet towel or other stimulus, or again, by insufflation with air.

The dilatation varies in extent. It depends upon the intolerance of the stomach and repeated vomiting, the resistance of the hypertrophied wall, the existence of bands or adhesions of the peritoneum holding down the stomach mechanically, extreme and prolonged reduction of food, and finally, upon the presence of tumor or induration.

Exploration with the Stomach Tube.—In a fasting stomach, anyone with persistent residual fluid and food debris in the early morning, has pyloric or subpyloric stenosis. Variations occur according to the amount of food, its quality, the state of the secretory apparatus and the degree of stenosis. One must guard the test by seeing to it that the proper test food and enough is given, the best of all being a mixed meal the night before. If the secretion be defective, the amount of fluid is less and the food will be coarser.

Severe cases with marked obstruction vary in symptoms according to (1) original cause and (2) rapidity of the course—slow or rapid.

(*a*) In slow course the stomach is tolerant, there is less vomiting with longer intervals between. Gastrectasis is extreme, the size of the stomach greater, more distended, the waves often very active; much fluid in the fasting stomach with food particles, fermentation, bad odor, perhaps blood, increased acid from organic acids; less commonly the acidity is from hydrochloric acid, free and combined, and acetic acid. Hydrochloric acid may be present even in cancer, when the digestive fluid of the test meal is strongly hypopeptic. The food taken several days previously is present, seeds of fruit, etc. It is hard to clean out completely by lavage. Tetany is common.

(*b*) In rapid course there is severe pain from contraction, the vomiting of small amounts and frequently because the stomach has not become used to the stenosis; rapid wasting and collapse; retention is less typical, as are also other signs, and the stomach is small.

Treatment.—The treatment is essentially *surgical,* though it must be confessed that a certain proportion of cases fail to receive the necessary relief. This, we believe, is due largely to faulty technique. Excision of the stenosed area or gastroenterostomy are the operations of choice according to the condition found. The reader is referred to surgical works on the subject.

Medical Treatment.—In benign cases of a mild type, the **nourishment** should be given in **small amounts**; food should be well divided, nutritious and easily digested. **Lavage** should be performed according to the requirements, in order to get rid of residue and to avoid the results of retention and fermentation. Patients with a definite stenosis, whether due to benign or malignant causes, should be urged to undergo surgical treatment as that is an inevitable necessity for a cure. Sometimes in severe cases, medical treatment suffices for a time, but no patient should be encouraged to undergo medical treatment for a long period of time. So long, however, as the weight is maintained and the patient is free from hemorrhage, severe pain or general weakness, medical treatment may temporarily give relief.

According to the degree of dilatation and the severity of the symptoms, **lavage** should be performed daily or every two or three days, according to the amount of retention. Not a few cases occur in which the dilatation is associated with the spasm caused by a duodenal ulcer, which does not obstruct, and in these cases medical treatment may be beneficial for a long period of time. In one case that the authors have seen, the patient showed marked evidence of stenosis, with visible peristalsis and 24-hour retention for two years before operation was performed, and during that period, the general health was maintained without any appreciable loss of weight or strength. At the operation, a nonobstructive duodenal ulcer was found, showing that the cause of the stenosis was due to spasm kept up through irritation of the distant ulcer. In these cases there is very often considerable acidity, for which Cohnheim and others recommend the use of **olive oil**, from 1 to 4 teaspoonfuls in the early morning. The effect of this is thought to be a lessening of the spasm and diminution of the acid secretion. When preferred a similar result may be obtained by the use of an emulsion of **oil of almonds**, or by the use of from 1 to 2 ounces of **fresh butter.**

Where we wish to get more of the constitutional effect, we can use some good iron preparation such as **syrupi ferri phosphate with quinin and strychnin compound**, which when given a teaspoonful before meals combines the local bitter effect with the constitutional. **Mixtures of iron, nux vomica** and **malt** are also very useful.

STENOSIS OF THE PYLORUS IN INFANTS

Synonyms.—Hypertrophic stenosis; pyloric stenosis of infants; congenital hypertrophic stenosis of the pylorus; pyloric disease (Génévrier).

The first accurate description of this disease is to be accredited to Beardsley in 1788, while the condition was brought to the general attention of the profession by Hirschsprung in 1888, who has given us the present conception of the condition.

Etiology.—The condition is more common in males and, according to some authors, more frequent in the artificially fed. The exact etiology of the condition is still undetermined, and whether or not a congenital hyertrophy of the circular muscles of the pylorus is the chief factor, or whether, on the other hand, there is a reflex or local pylorospasm due to ulcer, fissure or erosion, with secondary hypertrophy, remains in doubt.

THE CONGENITAL THEORY.—Doubt has existed as to whether the condition occurs in utero, although of recent years a few instances have been found in the foetus. Whether it is hereditary or whether it commences as a post-natal condition is equally undecided. Clinically, the first evidence occurs from within a few days after birth up to the seventh week, although why symptoms should not come earlier is not quite clear.

It should be remembered that in the new-born the pylorus normally is relatively thick, and later on becomes thinner. If the thickness of the pylorus persists, or if through the increased amount of food or curds the mucosa becomes thickened, it would be considered as pathological and would account for the development of symptoms after birth. Inflammation or irritation of a doubtful nature, affecting the young and very active cell structures, has been considered an etiological factor. Inflammation and hyperplasia would seem to predominate in the picture. Hypertrophy undoubtedly exists, whether from a continuous spasm or from some other cause. There is some neoplastic hypertrophy of the muscle coat, with hyperplasia of the connective tissue.

THE NEOPLASTIC THEORY.—There are those who consider the condition to be of the nature of a benign adenomyoma; certainly a tumor mass of muscle is sometimes found, very hard in consistence, sharply defined and covered with smooth peritoneum. Very often in these conditions no evidence of inflammation exists. The name "choristoma" has been applied to such a tumor. There is no relation between the size of the tumor and the age of the infant.

HYPERADRENALISM.—Pirie considers this a cause of pyloric hypertrophy by creating, first of all, pancreatic and pyloric insufficiency, which accentuate pyloric closure. J. Thomson's description is well worth quoting. According to him, the condition is due to "long-continued, inharmonious working of the various elements of muscular mechanism controlling the emptying of the stomach." It is difficult to determine which is the primary factor. It may be that an arrhythmia of the gastric contractions leads to disturbance of sphincteric action, but to what extent the combined nervous and anatomical factors work has not been determined. It would seem that in some way an abnormal local irritability of the infant's stomach is the primary etiological cause and creates a secondary hypertrophy of the annular muscle.

ANATOMICAL.—The average thickness of the wall in the infant is from 5 to 10 mm., while the average thickness of the muscle itself is from 4 to 7 mm. The main anomaly exists between the serosa and the submucosa. The latter is thickened; the mucosa itself is sometimes eroded, folded and shows ecchymoses. The duodenum is usually quite normal. The degree of dilatation in each case varies.

Symptomatology.—The symptoms may be described according to their stages. In the first stage, there are no symptoms, *i. e.,* there is a period when undoubtedly an anomaly exists prior to the development of clinical symptoms. The disease usually declares itself between the eighth day and eighth week. In this stage there is also hyperexcitability of the stomach with complete impermeability. Vomiting usually ushers in the symptoms and is a characteristic feature. It occurs shortly after nursing, after each, or only after several feedings. The vomiting is forceful and projective, the stomach contents being sometimes expelled several feet from the infant. The quantity, as a rule, is large and represents the entire feeding. On account of the frequency of the vomiting, the patient is sometimes persistently hungry, although not infrequently there is anorexia; the child's nutrition suffers; there is loss of weight, and often a rapid emaciation; weakness naturally supervenes. Later on, especially when dilatation occurs, there are signs of retention, the urine is diminished in quantity, the tissues are dry, there is constipation and evidence of toxemia. The epigastrium is prominent, visible peristalsis may be seen in the abdomen, and not infrequently a tumor is palpable, usually about the size of a walnut. The typical syndrome may occur without evidence otherwise of pyloric stenosis.

Palpation often reveals no tumor, or again, a tumor may be felt at one time and not again; or, as in Thorling's case, the tumor may be found in one position at one time, and later on, in a different situation.

The fluoroscopic screen often shows a typical picture; the cavity often enlarged, peristalsis is increased, and the stomach seems lobulated;

the waves are abnormally located in the upper pole of the stomach; the pyloric end is rounded. Later on, hypermotility is followed by fatigue of the muscle. Little or no food passes through the duodenum. The stools are usually pathological; there is evidence of mucus and bile, and there is often alternating diarrhœa and constipation.

Diagnosis.—This is based on the following points:

(1) The history, a healthy child vomiting a few days after birth, the emesis beginning suddenly and being projective in character.

(2) Loss of flesh, weakness.

(3) Evidence that food does not pass the pylorus into the duodenum. This evidence is general (thirst, constipation, diminished urine, dry skin) and local (visible peristalsis and, perhaps, a tumor).

As a rule, the x-rays are not required to demonstrate the condition. In fact, harm may often result if too much time and exposure is allowed —as in the case reported by Downes. In doubtful cases, however, a rapid examination by this means may often help to clarify the diagnosis. The same may be said of the use of the duodenal tube.

The condition differs from spasm, where delay is less marked and obstruction is intermittent. Sometimes the findings are negative; there may be no vomiting, no distention and no tumor. In Glover's case the first symptoms were a spitting of blood-tinged fluid the day after birth, persisting diarrhœa and anorexia, but no pain. The stools were green and yellow with fat curds.

Differential Diagnosis.—Here one must consider the following:

(1) The possibility of mere indigestion. One excludes the usual signs of stenosis.

(2) Mere pyloric spasm. Here the vomiting is less frequent and less projective. The stools are more or less fecal; there is less emaciation and constitutional disturbances, and there is no tumor.

(3) Congenital atresia of the duodenum. Atresia of the duodenum or congenital stenosis of the duodenum may simulate congenital pyloric stenosis. The patients, however, almost always live only a few days. If life is prolonged, there is recurrent vomiting every few days. The vomiting is usually bilious in character, though this depends on whether the obstruction is above or below the duct. As a rule, there are no stools other than mucus and cellular contents from the intestine. Inanition and wasting follow in these cases. The stomach and the first part of the duodenum usually becomes dilated; there is visible peristalsis, and sometimes one may feel a tumor-like mass. Many of these children are premature.

Treatment.—Treatment may be grouped under two heads, medical and surgical.

MEDICAL TREATMENT.—There are many who advocate medical treatment alone and say that operations are rarely necessary; nevertheless a continuance of medical treatment is warranted only if the loss of weight is less than 20 per cent. Furthermore, if after ten days, the treatment is unsatisfactory, operation is indicated. All in all, it is very doubtful if any medical treatment is as satisfactory as operative measures where a definite stenosis exists. Certainly the mortality has been so much diminished by surgical measures that it would seem to be the treatment of preference in the majority of cases. The usual medical treatment adopted in these cases consists in the following:

(1) **Gastric lavage** should be done twice a day, or as often as the case may indicate, using warm normal saline or a 1 per cent. sodium bicarbonate solution by means of a soft rubber catheter and funnel.

(2) **Feeding** is of importance. Breast feeding should be alternated with artificial feeding. Naturally, it is better, if possible, not to wean the child, but it may be found useful to use mother's milk artificially as well by means of a small medicine dropper. Feedings must be given slowly, and the milk from the artificial source should be relatively low in fat. Cow's milk is of doubtful value.

Levy advocates medical treatment and very little lavage. His formula is as follows:

Skimmed milk	10 oz.
Water	10 oz.
Farina	6 tablespoonfuls (level)
Dextrose	4 tablespoonfuls (level)

Maltose dextrin may be added if the dextrose tolerance is low. This formula may be weakened or strengthened according to need, but it is essential that the gruel should be thick. Feedings are given at intervals of three and a half hours, and after each one the child should be placed upon the right side to assist in the passage of the food.

Sauer also recommends **thick gruel** and claims to have cured 12 consecutive cases. His food consists of the following:

Farina	1 part
Milk	3 parts
Water	4 parts

This is boiled in a double boiler until the gruel adheres to the inverted spoon. Salt and sugar are added q. s. This feeding may be given alternately with breast feeding, if possible.

(3) **Enemata** should be given regularly, and after them, one may use **rectal injections** of saline or a glucose solution, 3 per cent.

(4) It is very doubtful if any drugs are of much avail. Alkalies, bismuth, gastric ferments, belladonna and atropin have all been employed with more or less success, as has also opium in small quantities. Bromids are probably useless. Of all the drugs that have been recommended, **atropin** is, perhaps, the only one from which beneficial results can be expected. Atropin is antispasmodic, as well as astringent, and has an inhibitory effect upon the gastric secretion. It can be given in the form of drops added to the feedings, from 2 to 10 drops of a 1-1,000 solution being used in the twenty-four hours. A good way to administer the drug is to dissolve an ordinary hypodermic tablet of 1/100 or 1/200 grain in the total quantity of food to be taken in the twenty-four hours. In this way, we can avoid the danger of using an inert preparation, the watery solutions deteriorating very quickly. A prescription recently recommended is as follows:

Neutral papaver hydrochlorid .010 (1-6 grain)
Glycerin 20. c.c. (5.42 fluidrams)
Aquæ bullientis q.s. ad. 100 c.c. (3.38 fluid ounces)
Sig.—1 to 5 drams (3.75 to 18.5 c.c.) two to four hours before meals.

Warm wet compresses over the abdomen may afford some relief, and should be applied with a binder.

If during the above method of treatment, the child does not lose any weight and the general symptoms are relieved, there should be no urgent recourse to surgical interference. If, on the other hand, despite the treatment, the weight suffers and the vomiting does not cease, **operation** is imperative.

Surgical Treatment.—The indications for surgical treatment are: (1) incorrigible vomiting, (2) absolute constipation, (3) poor general condition, and (4) x-ray evidence of obstruction. It is doubtful if incomplete permeability is a case for surgical interference. Operations should never be performed as an emergency. The patient should always be carefully prepared with lavage and infusion for at least four days if possible. Good technique is of paramount importance.

According to Gray and Reynolds, the essentials of the technique in operation are: (1) gentleness, (2) speed, (3) minimum manipulation, (4) pyloric incision as near the convex upper surface as possible, (5) division of the tumor along the whole length, and (6) control of the bleeding points with a few mattress sutures (gut).

The objections of operation are (*a*) that it enlarges the openings and (*b*) that it paralyzes the sphincter.

Of all the operations recommended, that ascribed severally to Fredet, Weber, Ramstedt, Keefe and others is to be commended most. The operation itself is of the simplest kind. It consists in a simple longitudinal incision through the serosa and muscularis of the pyloric ring, without sutures. The mortality is about 12.7 per cent.

Other operations are for the most part less commendable except under special conditions, *i. e.*, gastroenterostomy, pylorotomy or pyloroplastry. At the Boston Children's Hospital, 78 pyloroplastries have been performed, with 6 deaths.

The anesthetic and the anesthetist are of prime importance to a successful result. Ladd emphasizes the usefulness of introducing a catheter into the stomach during anesthesia. Gas oxygen is the anesthetic of election, according to some surgeons, while others prefer ether.

After operation, it is important to give enough water. **Ringer's solution**, 100 to 150 c.c., should be given twice daily by rectum or subcutaneously. The medical after-treatment and care in the feeding should be most strictly observed. **Breast-milk** with **buttermilk** should be given, one to two feedings from the bottle every two hours.

Duration and Prognosis.—On an average, untreated cases last four days, though the time varies from hours to weeks. The prognosis is grave unless treatment is instituted early, and with modern methods of treatment and improved technique the mortality has been greatly reduced. The outlook depends upon the duration of symptoms, the gravity of the general condition, the degree of the stenosis, efficient surgery and proper after care.

ACUTE DILATATION OF THE STOMACH AND DUODENUM

Acute dilatation of the stomach is a condition occurring most of all in young adults and is not uncommon. In one-half of the cases the duodenum is also dilated. Three groups may be established,—medical, surgical and obstetrical.

The *medical group* includes those cases which occur in acute infections or during convalescence, more especially in pneumonia, though cases have been reported during typhoid, peritonitis, scarlet-fever, etc. The condition likewise occurs in chronic diseases, pulmonary tuberculosis, cardiac diseases, nephritis, etc. Connor has drawn attention to its frequency in connection with deformities of the spine, and Neck has described one case following the use of 50 centigrams veronal. Cases are described as following mere overloading of the stomach (*paresis ab ingesta*).

Surgical cases have followed trauma, contusions of the abdomen, and some cases have been described as occurring after the application of plaster jackets. Most frequently, however, they have followed abdominal operations, especially on the stomach and bile passages, so much so that the term "post-operative acute dilatation" has been applied. It is extremeley doubtful it this term is justifiable, all the more so as the anesthesic may be the main factor in most of the cases. Chloroform has been the most commonly used narcotic in these cases. Nor does the condition necessarily follow the opening of the peritoneum, as it is frequently seen with nephropexia and after gynecological operations.

In *obstetrical cases* it is more rare, and appears to follow the long use of chloroform, especially in long forceps operation.

Pathogenesis.—(1) It is doubtful if the extrinsic nerves have any influence on the genesis of acute dilatation. Alvarez, Carlson and others have shown that a peripheral automatic mechanism exists in the gastrointestinal organ, and that a division of the extrinsic nerves is unimportant in its effects upon this. Gastric tone is the more imporant factor. Absorption of toxic products has little to do with the pathogenesis.

(2) Kinking of the duodenum or constriction by tense mesentery. It is well known that the third part of the duodenum may be easily compressed by traction of the mesentery and its vessel against the aorta and the vertebral column. Some consider this as the primary condition, while others regard it as secondary, *viz.*, a post-operative acute paralytic atony, in which the large stomach thus dilated pulls upon the mesentery, thus bringing about the condition.

Borchgrevink concludes that the first event is the dilatation of the stomach; that mechanical occulsion of the duodenum is caused either by direct pressure of the stomach on the duodenum as it crosses the spine, or by secondary arterial mesenteric compression, this latter being brought about by traction of the small intestines which have been forced into the pelvis.

Dragstedt considers the dilatation as the primary factor and the toxic symptoms as being due to duodenal stricture.

In discussing etiology, one may say that one chief feature is common to the surgical group, *viz.*, the use of general anesthesia, usually chloroform. The doubt that exists has reference to the relation between cause and effect, pathogenesis and the relation to the other phenomena, *i. e.*, secretion, dehydration, collapse and death.

Symptomatology.—The condition is usually ushered in with symptoms that suggest a serious abdominal complication. There is often severe epigastric pain, either well localized or extending to the left hypochondrium. Following upon this, there is uncontrollable vomiting of much fluid, often foul-smelling though not fecal in character. It contains little or no hydrochloric acid, though always bile, and often pancreatic juice and duodenal secretion. The colour is brownish, sometimes almost black. This large amount of fluid would seem to arise from some abnormal stimulus, chemical rather than nervous, and is of the nature of a huge secretion into the upper intestine and stomach. Dehydration, with its attendant symptoms, accompanies the picture. It may be that the toxic substance (secretagogues) which enters the blood stream brings on the secretion of fluid. The toxemia is due to the absorption, not from the dilated stomach, but from the occluded duodenum, and adds to the gravity of the picture. Unless relief is afforded early by suitable treatment collapse supervenes.

Physical Examination.—On inspection, the abdomen is markedly distended, especially the epigastrium. One can often see the outline of the bulging stomach, and sometimes with a large weight of fluid the viscus can be seen to change its position according to the posture of the patient. On palpation one can readily feel the full stomach and obtain fluctuation on tapping with the finger.

The presence of succussion is only of relative importance, and is not pathogonomic of dilatation. Percussion yields an extensive area of dullness by means of which one may easily delimit the area of the stomach. Apart from these signs, there is not much of diagnostic importance on inspection. There is no rigidity, nor is there visible peristalsis. The picture is often confused with intestinal obstruction. Passage of the stomach tube and removal of the fluid rapidly alters the clinical picture and the physical signs, as well as affording great relief to the patient. The stomach, however, rapidly fills again, and the grave signs that ushered in the disease return. Not uncommonly tetany occurs, probably due to the excess of absorption of intestinal poisons to such an extent that the parathyroid glands have no control. (The theory that tetany is due to reflex irritation from the stomach or intestines is no longer tenable.)

Diagnosis.—The diagnosis is made from the rapid onset of the clinical signs above given and the fact that they usually follow in the wake of some serious acute disease or an operation performed under a general anesthetic.

In the *differential diagnosis,* one must consider (1) post-anesthetic vomiting, (2) intestinal obstruction (fecal vomiting), (3) perforative peritonitis, (4) acute pancreatitis, and (5) uremia.

Careful attention to the symptoms and physical signs of the enlarged stomach filled with fluid and the vomiting of large quantities of fluid of a dark brown colour, and the relief by proper lavage, constitute the main features in the differential diagnosis.

Treatment.—Prior to rational therapy the mortality was high. To-day the outlook is much less grave and the number of cures relatively large. The essentials in the treatment follow:

FOOD AND DRINK.—All foods and drink should be withheld.

THOROUGH EMPTYING OF THE STOMACH BY MEANS OF PERSISTENT LAVAGE WITH THE STOMACH TUBE.—For emptying the stomach, one may use plain water or normal saline solution, or a deci-normal solution of bicarbonate of soda. The removal of fluid relieves the pressure on the over-filled stomach and the adjacent parts, and lessens the tendency to occlusion of the duodenum. It encourages peristalsis and minimizes any possible cause of toxemia. The lavage must be often repeated, at first every two hours according to requirements, or as often as the stomach seems to fill. Bassler and others recommend continuous lavage. Lyons has devised a method of **continuous lavage** by means of the duodenal tube, the end being kept in place by means of adhesive strapping attached to the cheek. A glass T-tube is inserted into the duodenal tube, and by means of irrigation from a tank over the head of the patient and connected to a collecting basin below, the fluid is continuously siphoned off.

POSTURAL TREATMENT.—Various **postures** are recommended to encourage the emptying of the stomach and relieve pressure. The **foot** of the **bed** is **elevated** and the patient is placed either in the anterolateral position of the abdomen, or where possible, in the knee and chest position. This postural treatment is intended to release mesenterical occlusion of the duodenum by removing the downward traction of the mesentery, which, in turn, is due to the weight of the over-pulled stomach.

SALINES.—Administration of **saline** is recommended either intravenously (salt or Ringer's solution), or by hypodermoclysis or proctoclysis. This relieves the thirst and the weakness, and supplies the tissues with much needed fluid.

STIMULANTS.—**Stimulants** should be given, hot water bottles applied, and proper nursing provided.

DRUGS.—It is doubtful if any special drug treatment is of much avail. Muscle stimulants are recommended, such for example, as a solution of **pituitary body,** 15 minims (.92 c.c.) or **strychnin sulphate** or **physostigmin sulphate,** 1/50 grain (.00043 gram). Doses of **atropin** are also doubtless of some use, though eserin is not to be relied upon.

OPERATION.—Operation has not proved successful hitherto. One must differentiate between necessity for operation in intestinal obstruction, which is urgent, and in acute **gastrectasis**, where it is unwise.

ATRESIA OR STENOSIS OF THE DUODENUM

This is a rather infrequent condition, usually congenital, cases of which have been reported in the literature since 1803. It is met with in infants, and usually results in death in from a few hours to a few days.

The condition begins in the fourth week of foetal life. At that time the epithelium of the duodenum proliferates to such an extent that it fills up the lumen of the gut; later this epithelial plug becomes absorbed or canalised and it is the failure of this latter process to take place properly that results in either atresia or stenosis of the gut.

Types.—Three types occur as follows:

Complete Atresia.—If canalization does not take place at all, we have the proximal and distal portions of the gut united by a fibrous cord.

Partial Atresia.—If the process fails before its normal completion, we have a narrowed lumen and a stenosed portion of the gut resulting.

Occlusion by a Diaphragm.—If again absorption of the plug only just falls short of its normal completion, we may have in a normal appearing gut a diaphragm or septum persisting. This diaphragm is situated about the papilla of Vater, usually just above it, and may or may not be perforated. When one considers that the liver buds out from the anterior wall of the duodenum about the fourth week, and the pancreas from the posterior wall about the same time, it is easy to imagine that many malformations may happen in this area of great specialization.

Cautley in 1919 collected all the cases of atresia and stenosis of the duodenum which had been reported in the literature. These, with a case of his own, made in all about 100 cases, and several others have been reported since. He found that atresia was more common than stenosis and that occlusion by a diaphragm was very rare. In 48 cases of atresia, life lasted from 30 hours to 9 days; in 9 cases of stenosis from 30 hours to 14 days. In Cautley's own case of stenosis, the child lived for 13 months and 1 week; and he cites a remarkable case, reported by Harrison, of complete occlusion of the duodenum by a diaphragm situated opposite the papilla, where the child lived for 9 months. The specimen from this case is said to be in the museum of St. Barthlomew's Hospital. Other types are acquired. In addition to developmental defects, there are several other less frequent causes of duodenal stenosis which have been noted from time to time, such as pressure of the head of the pancreas, antenatal peritonitis, probably syphilitic in origin, and absence of the pancreatic duodenal artery. When due to peritonitis, the obstruction is generally situated nearer the duodenojejunal junction, and there is fibrosis around the point of constriction.

Symptomatology.—The symptoms are vomiting and constipation, and Cautley says that in 90 per cent. of cases, the vomiting contains bile. If life is prolonged, we get the signs of obstruction and visible gastric peristalsis, and the development of a palpable mass caused by the dilated proximal portion of the duodenum.

The points of difference between this condition and congenital pyloric stenosis are that the vomitus frequently contains bile and that the contour of the abdominal tumor is less clearly outlined. Even where the obstruction is above the papilla of Vater, we may get bile in the vomitus as the common duct often has a branch which opens into the duodenum *above* the obstruction.

Treatment.—The treatment of the condition is surgical only; few cases recover; obviously an early diagnosis and a skillful surgeon are essential. Cautley points out that in the cases collected by him, 5 per cent. presented other developmental anomalies as well, such as imperforate anus, absence of organs, or atresia of the esophagus, urethra or rectum, so that very often all treatment is of no avail.

There have been a number of cases reported of chronic duodenal obstruction with dilatation occurring in older children and adults. In these cases the obstruction is not due to developmental errors, but to some other cause such as pressure from enlarged retroperitoneal glands, tumor of the head of the pancreas, or the peritoneal adhesions resulting from ulcer or other causes. Dilatation of the duodenum is also often present when we have marked gastric dilatation (*v. antea*).

Roberts reports the case of a man of forty-two years old, who had had abdominal pain and vomiting since childhood. A barium meal passed through the stomach in normal time, but the duodenum was shown as an enormous C-shaped sac with an air bubble on top. Operation showed a cicatrical band at the duodenojejunal junction, and a gastro-enterostomy gave relief.

Chronic obstruction may also result from pressure on the duodenum by the superior mesenteric artery. In those cases we get all the signs of obstruction but when the abdomen is opened and the mesentery raised the condition is immediately relieved.

Meyer reports two such cases, and Reinhard says that obstructions of this kind can be relieved by posturing, and that when this does not suffice, the abdomen should be opened and the intestine pulled out of the pelvis.

Scrimger reports a case of this kind which occurred in the surgical service of the Royal Victoria Hospital. The patient was a mentally deficient child of eleven years, who had suffered from frequent vomiting since early infancy; sometimes the vomitus contained food taken two or three days before. When first seen, the child was small, poorly-developed, and weighed only 55 pounds. The abdomen was moderately full, and there was strong visible gastric peristalsis. The skiagrams showed the stomach to be moderately dilated and the duodenum very much dilated. The abdomen was opened and the stomach and pylorus found to be normal. The duodenum was greatly enlarged and the rest of the bowel was normal. The obstruction of the duodenum was at the point of crossing of the superior mesenteric artery and was relieved by lifting up the mesentery. An anastomosis was made between the second part of the dilated duodenum and the first part of the jejunum, and the child enjoyed an uninterrupted recovery. She left the hospital one month later in good condition, and after eight months, had had no vomiting, had grown four inches and had gained 14 pounds in weight.

In all these cases of chronic obstruction, posturing and lavage as recommended by Reinhard should be tried: Bassler says that he has seen only one case of duodenal obstruction caused by bands, kinks or angulation, but when these measures fail to give relief, the abdomen should be opened and the proper surgical measures carried out.

CHAPTER XVIII

FUNCTIONAL DISORDERS OF THE STOMACH

By George Morris Piersol, M.D.

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Definition.—Functional disorders of the stomach, or the so-called gastric neuroses, represent those conditions in which perversions or alterations in the functional activities of the stomach occur without the presence of any demonstrable lesion of that organ. It must be clearly borne in mind that no condition can properly be regarded as a neurosis or pure functional disturbance in the presence of any organic gastric condition.

A generation ago considerable space was devoted to the gastric neuroses in all the systematic works upon diseases of the stomach. It is noteworthy that in recent years in such books the number of pages devoted to the consideration of functional disturbances of the stomach has steadily diminished. Improvements in diagnostic technic, the work of the skiagraphers, but particularly the observations of the abdominal surgeons, have clearly shown that many of the disorders that at one time were regarded as functional are in reality but manifestations of some definite organic disease. With the present steady increase in our knowledge, doubtless many conditions looked upon today as functional in character will ultimately be shown to be organic in their nature. Nevertheless, there are, and probably always will be a certain number of gastric disturbances that justifiably may be looked upon as primarily functional in character.

Classification.—It has been customary to group the gastric neuroses under two main etiological heads, the primary and the secondary. By the primary are meant those conditions in which the seat of the disturbance resides entirely within the stomach. The secondary group embraces those functional disturbances of the stomach which are the result of some underlying disturbance elsewhere in the body. This latter group is by far the more important, and includes that large number of instances in which gastric disturbances are secondary to disease of other abdominal organs, such as the gall-bladder, appendix, pancreas, liver, colon, or the pelvic organs. In other instances the gastric disturbances are secondary to disease of organs more remote from the stomach, such as the central nervous system, the thyroid, the heart, the lungs, and the blood-vessels. Many gastric neuroses are secondary to functional disturbances of the nervous system, as observed in hysteria, neurasthenia and the various psychoneuroses, and finally there may be secondary derangements of gastric function, the result of constitutional and chronic visceral disease such as diabetes, gout, the anemias and nephritis. The primary group of gastric neuroses is a comparatively small one, and although in this class the disturbance appears to lie in the stomach, it has been shown that in almost every instance some functional disorder of the nervous system is actually the underlying cause. In reality, therefore, all gastric neuroses are, strictly speaking, secondary, and are but manifestations either of some disorder of organs lying adjacent to or remote from the stomach, or of some functional or organic nervous disturbances or of some constitutional condition. From the standpoint of both diagnosis and management, it is important to bear in mind such a conception of the gastric neuroses, since it often happens that gastric symptoms so dominate the clinical picture, that their relationship to other disease conditions is lost sight of.

Gastric Function.—Before taking up the clinical aspect of functional disturbances of the stomach, it will be well to review briefly certain essential facts concerning gastric functions. By far the most important functions of the stomach are motor. The movements of the stomach consist of waves of peristalsis, having their origin at the cardiac end of the stomach and sweeping on towards the pylorus. There is relatively little movement at the fundus of the stomach where the food that has been ingested lies undisturbed, thus permitting the continued action of the ptyalin derived from the salivary secretion. The peristaltic waves become much more active in the body of the stomach, and increase in their intensity as they progress toward the pylorus. These gastric movements propel the food forward and at the same time allow fresh masses of food to come in contact with the mucous membrane, thereby bringing about a proper admixture with the hydrochloric acid and pepsin.

Alvarez has pointed out that the nonstriated muscle of the stomach, as well as that of other portions of the digestive tract, is to a great extent, autonomous and in itself possesses the property of initiating peristaltic movements. The same author believes that the pace-maker of the stomach, as it were, is that part of the lesser curvature that lies close to the cardia, and that from this point down to the pylorus there are gradients of rhythmicity, tone, irritability and metabolic rate, and that these gradients are higher nearest the cardiac end and lower towards the pylorus. It is this variation in the gradients of the different portions of the intestinal tract including the stomach that, according to Alvarez, is chiefly responsible for the onward and downward direction of the gastric movements. A variation in one or several of these gradients may so alter the normal course and direction of the gastric movements as to create clinical symptoms.

The result of the motor function of the stomach is to expel the acid chyme through the pylorus into the duodenum. Cannon demonstrated that an important factor in regulating the pyloric mechanism, by which only that amount of acid chyme is allowed to enter the duodenum which can be properly taken care of by the digestive juices of the intestine, is the degree of acidity that exists on the gastric or duodenal side of the pyloric sphincter. A certain degree of acidity on the gastric side brings about relaxation of the pylorus, and when the acidity on the duodenal side reaches a certain degree, the pylorus is closed. In addition to this chemical regulation of the pyloric sphincter, it seems likely, from the work of Alvarez and others, that the opening and closing of the pylorus is also dependent upon the differences in the gradients of tone and irritability that exist between the stomach and the first part of the small intestines. These motor functions are here emphasized because they play a dominating rôle in gastric physiology and the modifications which they undergo in abnormal states are largely responsible for many gastric symptoms.

The extrinsic nervous mechanism of the stomach, supplied through the vagus and abdominal sympathetic, is of undeniable importance in regulating the motor function of the stomach; although, contrary to the belief of some of the older physiologists, it is not essential for gastric movement, since as has been above pointed out, like other smooth muscle, the gastric musculature is autonomous. Both in health and disease nerve impulses play an important rôle in regulating gastric peristalsis. The plexus of Auerbach, lying between the longitudinal and circular layers of gastric muscle, serves to bring the musculature of the stomach under the control of impulses conveyed through the vagus and sympathetic trunks. Alvarez believes that the function of Auerbach's plexus is to conduct stimuli in order to coördinate movements of the gastric musculature, to make the muscles of the stomach respond to the stimuli that come from the underlying mucous membrane, and further to prevent the gastric muscle from being too active and contracting down into a hard knot. The extrinsic nerves of the stomach are capable of stimulating or depressing the organ either as a whole or in part, and in this way probably the nervous mechanism is capable of bringing about alterations in the irritability and tone of the gastric musculature.

Although recognizing the physiological importance of the autonomy of the gastric muscle, one must not lose sight of the important rôle that the extrinsic nervous mechanism of the stomach plays in bringing about functional disorders of that organ. The nerves that supply the stomach are a part of the vegetative or autonomic nervous system, which is made up of two great groups of fibers, those which originate from the sympathetic proper, and those which are a part of the great vagus system. In a general way it may be said that the function of the vagus is to stimulate, that of the sympathetic to depress. Eppinger and Hess have pointed out, largely on theoretical grounds, that under certain conditions the tonus, or excitability, of one or the other divisions of the autonomic nervous system may become so altered as to disturb the normal balance so that either the vagus or sympathetic influence dominates. When by reason of increased tonus the vagus system dominates, they have termed the condition vagotonia. When the opposite is true, and the sympathetic nervous system is in the ascendancy, the condition has been termed sympatheticotonia. Inasmuch as the nervous control of the stomach resides in the autonomic nervous system, when either of these conditions, vagotonia or sympatheticotonia exists, it is easy to conceive of modifications of normal gastric functions, the result of alterations in the tonus of either of the two great divisions of the autonomic nervous system. It is more than likely, therefore, that functional disorders of the stomach are in many instances merely local manifestations of these vagotonic or sympatheticotonic states. So far as the stomach is concerned, it is said that increased vagal tone brings about increased or even reversed gastric peristalsis, spasm of the cardiac and pyloric sphincters and increased gastric secretion. On the other hand, sympatheticotonia may be responsible for diminished gastric secretion, atony of the gastric musculature and defective gastric peristalsis.

The chemical function of the stomach is of less importance than the motor, since for the most part digestion is carried on in the small intestine. The gastric juice, the active principles of which are hydrochloric acid and pepsin, begins to flow as the result of nervous stimuli that are initiated by the pleasure of seeing, smelling and taking food. The first flow of gastric juice, referred to as the appetite juice, acts as a stimulant to further gastric secretion. Carlson's observations would indicate that in an ordinary individual, after a mixed meal, the total amount of gastric juice usually secreted, amounts to 700 c.c. The same observer has also shown that even when no food is taken there is a fairly continuous secretion of gastric juice, varying in amount from a few cubic centimeters up to 60 c.c.

Practically no absorption takes place through the stomach. Although water and the ordinary products of digestion are not absorbed, hydrolyzed protein and concentrated glucose solution are taken up.

Although frequently various subjective sensations, including pain, are referred to the stomach, it has been definitely shown by numerous observers, that the stomach along with other viscera, is insensitive to all forms of painful stimulation. The explanation of the pain felt in gastric disturbances is found in the "viscerosensory reflex" so aptly described by Sir James Mackenzie. According to this theory, when a viscus incapable in itself of painful sensation, is abnormally stimulated by some morbid process, unusually strong impulses are conducted along the sympathetic nerves running from the viscus to the spinal cord. These abnormal impulses have an effect upon adjacent ganglion cells in the cord. If these adjacent cells in the cord are sensory, when thus stimulated they convey afferent impulse to the brain which are recognized as pain and are promptly referred to the peripheral distribution of the sensory nerves involved. Thus, abnormal stimuli arising in the stomach are conveyed by afferent sympathetic nerves to those segments of the spinal cord that supply certain portions of the abdominal wall, where the pain and discomfort of gastric disorders are felt. In the production of pain and other sensory disturbances which have their origin in the stomach, the importance of the motor functions of the stomach is again apparent, since the stimuli which give rise to these sensations are in all probability really the result of increased tension of the gastric wall or abnormal or violent contractions of its musculature.

For the sake of convenience the clinical manifestations of functional disorders of the stomach may be considered under three main heads, according to whether motor, secretory or sensory disturbances dominate the picture. It should be clearly understood, however, that although at times disturbances involving only one important gastric function are encountered, nevertheless it is not infrequent to meet instances in which several functions are equally disturbed. Furthermore, emphasis must again be placed upon the fact that although these functional disturbances may occur as pure neuroses, any or all of them are frequently encountered associated with organic disease of the stomach, in which case they must not be looked upon in the light of a neurosis but rather should be regarded as part of the symptomatology of the underlying anatomical lesion.

Etiology.—There are certain etiological factors that are common to practically all functional disturbances of the stomach. As a rule disorders of this character are more common in women than in men, although in private city practice, where a large number of men are leading their lives under tension, the disproportion between the two sexes is not great and a surprisingly large number of men suffer from gastric neuroses. Functional disturbances of the stomach rarely occur in the very young, and are uncommon in old people. They occur chiefly between the ages of eighteen and forty-five, the largest number being observed between the twenty-fifth and thirty-fifth years. As might be expected, gastric neuroses are more common in those races, as for example the Polish Jews, that are particularly susceptible to functional nervous disorders. The upper classes are more often afflicted with gastric neuroses than the lower classes. They occur more frequently in those who lead confined lives, who exercise too little and eat and drink too much, especially in those who live at a high tension, and are constantly subjected to mental overwork and worry, and who are given to excesses whether business, professional or social. Emotional strains of all kinds are frequently determining factors. Over-indulgence in alcohol, tobacco, tea and coffee, rapid eating, irregular meals, and the habitual use of highly seasoned, rich and irritating food, may all be the exciting causes of functional gastric disturbances. In women, it is not uncommon to see gastric neuroses develop as the result of menstrual disturbances and pregnancy. It is rare to find gastric neuroses in those whose nervous systems are stable. They are usually found in individuals who have an underlying neurotic, high strung, emotional, neurasthenic or psychasthenic nervous system.

There seems to be no great unanimity of opinion as to the frequency with which functional disorders of the stomach are encountered. They are certainly much more uncommon in the wards and out-patient departments of general hospitals than in private practice. It has been estimated that about from 3 to 5 per cent. of gastro-intestinal cases coming into a general hospital, can be regarded as instances of gastric neuroses. On the other hand, in private practice, various observers estimate that gastric neuroses comprise anywhere from 13 to 55 per cent. of all gastric cases. The latter figure is far in excess of that generally given. From 15 to 20 per cent. is a fair average of the number of private cases of gastric disturbances that can be looked upon with reasonable certainty as instances of gastric neuroses.

Symptomatology.—There are certain symptoms that are more or less characteristic of all forms of gastric neurosis. There is no regularity in the way in which the symptoms develop. In some instances the onset is acute and no exciting cause can be discovered for the condition. On the other hand, the symptoms may be gradual in onset and slowly progressive. Functional disorders of the stomach run no definite course. intensity of the symptoms varies markedly from time to time. They may cease with surprising abruptness and for no discoverable cause, only to recur with equal suddenness. The multiplicity and variability of the symptoms is characteristic of gastric neuroses. Unlike organic disease of the stomach, certain definite symptoms are not prone to persist in functional disturbances. There may be marked variation in the symptoms from week to week. The fact that the severity of the symptoms bears no relationship to the kind of food, or to the taking of food is equally suggestive. In many cases of gastric neuroses, the most extraordinary combinations of indigestible food can be taken with impunity, whereas the simpler forms of nourishment may bring on a train of most distressing symptoms. The symptoms may occur at any time and fail to bear a definite relation to the taking of food, such as is usually observed when the cause of the digestive disturbance is organic.

Gastric symptoms are frequently associated with nervous symptoms of a general character, such as depression amounting to melancholia at times; irritability; insomnia; hysteria and various psychoneurotic states. It is not infrequent for those who suffer from gastric neuroses to exhibit areas of cutaneous hyperesthesia and even anesthesia, and the gastric symptoms are frequently aggravated by emotional and nervous disturbances. Patients who suffer from gastric neuroses, may be entirely free of symptoms until they are subjected to some unusual strain, worry, or disappointment following which the gastric symptoms become pronounced. When pain is present it is rarely localized, and is not associated with rigidity and tenderness. It is more apt to be vague and diffuse. A large number of ill-defined subjective sensations are often complained of. Decided variability in the gastric analysis, hyperacidity alternating with subacidity, or an absolutely normal gastric analysis is not infrequently encountered. In the early stages of functional disorders of the stomach, there is rarely any noteworthy disturbance in the nutrition. As the disease progresses, the dietetic restrictions which these patients are apt to impose upon themselves, frequently lead to marked under-nutrition, so that in long-continued cases of gastric neuroses, it is not uncommon to find them complicated by secondary anemias and malnutrition. Careful analysis of the symptomatology must lead one to the conviction that in all true gastric neuroses the symptoms referable to the stomach are but the local manifestations of a general functional disorder of the nervous system.

Diagnosis.—The diagnosis of functional disturbances of the stomach should never be made until all possibility of organic disease, either of the stomach or in organs remote from the stomach, has been definitely excluded. At best this is no easy task, because of the frequency with which functional disturbances of the stomach are associated with or develop in the course of organic disease. The frequency with which functional conditions of the stomach are associated with organic disease remote from that organ, as for example in the course of organic disease of the central nervous system, or of the lungs, or of the heart, must never be lost sight of. It is particularly important to exclude disease of other abdominal organs, since it is a common occurrence to find functional gastric disorders masking some underlying causative condition, such as chronic cholecystitis, with or without stone; a chronic appendix; disease of the liver or pancreas; colonic stasis or visceroptosis. In order to exclude all possibility of organic disease, every case of suspected functional disturbance of the stomach should receive a thorough physical examination, and if the symptoms persist such examination should be frequently repeated. Careful investigation of the urine and of the blood should never be omitted, and in every instance repeated gastric analyses, with a view to testing the secretory and motor functions of the stomach, must be resorted to. An additional safeguard is a properly carried out roentgenological examination of the gastro-intestinal tract.

The occurrence of certain symptoms should always make one hesitate to make the diagnosis of a functional gastric disturbance. Some of these which may be mentioned are persistent and localized abdominal pain; the occurrence of even moderate degrees of gastric retention; persistent and high degrees of gastric hyperacidity, especially when associated with gastric pain and heart-burn; the occurrence of pain in the upper abdomen at definite times after eating; the existence of a gastric subacidity, associated with pain, vomiting or retention, the association of gastric symptoms with anemia and progressive loss of weight and strength; and the occurrence of blood in the stools, occult or macroscopic, unless some obvious local cause for the bleeding can be found. It should also be borne in mind that just because a nervous individual suffers from gastric disturbances, there is no reason why that individual may not also be the subject of definite organic disease. Finally, the fact that gastric neuroses rarely develop in people over forty-five should make one extremely cautious in making such a diagnosis in any except younger individuals. It cannot be emphasized too strongly that the diagnosis of a gastric neuroses should never be entered into lightly. It is justifiable only after all possible organic disease of the stomach or of other organs has been most carefully excluded.

DISTURBANCES OF MOTOR FUNCTION

PERISTALTIC UNREST

By this term is meant abnormally violent peristaltic movements of the stomach which are usually more or less visible. These violent peristaltic movements as a rule take their origin near the fundus of the stomach and sweep towards the pylorus. At times antiperistaltic movements have also been observed.

Etiology.—Generally, excessive peristalsis cannot be looked upon as a neurosis, since it is associated with varying degrees of pyloric obstruction. Occasionally however, it may be observed in neurotic and neurasthenic individuals, and when no pyloric obstruction is present the condition may be regarded as a pure neurosis.

Symptomatology.—In addition to the general symptoms of the associated or underlying functional nervous disturbances, patients who suffer from peristaltic unrest complain of uncomfortable sensations in the region of the stomach, the result of the excessive or abnormal muscular contractions. Actual pain is rarely present. From time to time other symptoms such as nausea, belching or sour eructations may be complained of.

Diagnosis.—The raritv of peristaltic unrest as a pure neurosis should be borne in mind in making a diagnosis. The important step in such a diagnosis is to exclude by every possible means the presence of any organic obstruction of the pylorus, or of a pylorospasm secondary to disease of the other abdominal organs.

Treatment.—There is no special local treatment of the condition, although the application of the **faradic current** both **externally** and **internally** has been recommended. In common with other gastric neuroses, the essential feature of the treatment is to improve the general condition of the patient, and to correct the underlying functional disorders of the nervous system.

Prognosis.—The prognosis is good if the underlying nervous conditions can be removed.

HYPERMOTILITY

Gastric hypermotility must not be regarded as synonymous with peristaltic unrest. The former is rarely if ever a neurosis, whereas the latter may be. In hypermotility increased peristaltis of the stomach is associated with the rapid emptying of chyme into the small intestines. Hypermotility usually occurs in association with achylia gastrica; it sometimes accompanies diarrheal conditions, and is even observed in carcinoma of the stomach and chronic duodenal ulcer when the pylorus is relaxed and non-obstructed. The rapid emptying of the stomach in achylia, when the normal acid reaction that is required to open the pylorus is lacking, is a phenomenon which seems to be at variance with Cannon's conception of the chemical mechanism responsible for opening and closing the pyloric sphincter. That rapid emptying does frequently occur with achylia, is a well demonstrated clinical fact and the explanation is probably to be found in Alvarez' theory of gradients of tone and irritability which are graded downward from the cardiac end of the stomach to the pylorus.

MERYCISM OR RUMINATION

Rumination is a condition in which small portions of food are regurgitated into the mouth, either to be ejected or again masticated and swallowed. The exact mechanism by which rumination occurs is not clearly understood. By some it is thought to be purely the result of relaxation of the cardia while by others it is looked upon as a result of irritation of the vagus which causes dilatation of the cardia and antiperistaltic waves in the esophagus. Dilatation of the lower end of the esophagus has also been held responsible for the condition.

Etiology.—Rumination is found more often in men than in women. It seems to be more common in the intellectual classes than in those that are not brain workers. It occurs more often in high strung, neurotic individuals than in those that are nervously stable. Age seems to have no etiological bearing on the condition. In some cases the onset appears to have been due to nervous shock and worry. A number of observers believe that heredity plays a certain part in causing a predisposition to the affection. It is also said that the habit may be acquired by imitation.

Symptomatology.—The onset of rumination is usually gradual. It apparently begins with the regurgitation of food. As a result of this regurgitation, a certain agreeable sensation is experienced. Gradually the patient begins to reswallow the regurgitated material, until finally a habit is established which ultimately becomes involuntary. Regurgitation may take place after every meal, or only occasionally. It usually occurs soon after the meal, but may be delayed for some hours. It may last for a long or a short time after a meal. When regurgitation or rumination takes place soon after the ingestion of a meal, the material regurgitated retains the taste of the original food; when rumination occurs some hours after a meal, the regurgitated material is usually bitter or sour. Rumination is not accompanied by vomiting or nausea. It more often occurs alone but may be associated with other gastric symptoms. The gastric analysis is as a rule normal and the nutrition of the patient is not affected. Sometimes the condition disappears spontaneously, only to recur when the individual is subjected to some unusual nervous strain.

Diagnosis.—The diagnosis is readily made by the fact that the regurgitated food is remasticated, and the regurgitation is not associated with nausea or vomiting, and is accompanied by a distinct pleasurable sensation.

Treatment.—The essential thing in the treatment of rumination is **auto-suppression.** The condition is often relieved by teaching the patient to masticate thoroughly and to chew slowly. Any abnormalities of the gastric secretion should be corrected, and the general condition of the patient should be improved in every possible way. Strychnin and quinin have both been given; they have no specific action, but owing to their bitter taste, tend to lessen the desire to ruminate.

Prognosis.—The prognosis is as a rule good, especially if the patient can be made to realize that rumination is but a bad habit and is largely under the control of the individual's will.

REGURGITATION

Regurgitation, the bringing up of small quantities of food from the stomach into the mouth whence they are ejected, sometimes occurs as a pure neurosis, but it is frequently associated with organic disease of the stomach, such as the various forms of gastritis, gastric dilatation, gastric ulcers and neoplasms. When regurgitation occurs as a pure neurosis, it is usually associated with nervous excitements, strains, shocks and worries.

Symptomatology.—When regurgitation is a true neurosis, the act is involuntary and usually continues throughout the entire period of digestion. The longer after the ingestion of food that regurgitation takes place, the more acid is the regurgitated material. In some individuals regurgitation only occurs after certain meals or after the ingestion of special types of food. When regurgitation is persistent and severe, there may ultimately occur some impairment of the general nutrition. When it is a pure neurosis, there are no noteworthy changes in the gastric analysis.

Diagnosis.—Regurgitation must be differentiated from rumination and from vomiting. As above pointed out in rumination the food is remasticated and reswallowed; in regurgitation it is ejected. Vomiting is accompanied by the well-known manifestations of nausea and retching, which are absent in regurgitation. The essential thing in the diagnosis is to eliminate the possibility of organic disease of the stomach before regarding regurgitation as a pure neurosis.

Treatment.—The treatment consists chiefly in **psychotherapy** with **autosuppression** as its object. The general nervous disturbance must be carefully managed. If the general health of the patient has suffered, forced feeding and rest frequently must be resorted to.

AEROPHAGIA OR ERUCTATIO NERVOSA

By aërophagia is meant periodic attacks of noisy belching. At one time it was thought that the gas thus expelled was due to fermentation and had its origin in the stomach, a belief which is fondly cherished by most sufferers from this condition. It has been shown, however, that this is not the case, but that in reality the gas which is belched with such noise and force is but swallowed air. Under normal conditions small quantities of air are swallowed when food is taken. In aërophagia, voluntary, violent deglutition forces the air into the esophagus and stomach; sometimes the swallowed air never gets further down than the esophagus, and it is then expelled by forcible contractions of the esophagus. Such expulsion is a voluntary act, and is indulged in to relieve various uncomfortable sensations that are referred to the stomach. Aërophagia must be regarded in almost every instance as a primary neurosis.

Symptomatology.—It occurs chiefly in young people, and in those who are neurotic and emotional. The onset sometimes follows some unusual care, worry or nervous excitement. The ability to swallow and expel air forcibly may easily be acquired and is often indulged in by children as a form of amusement. The expulsion of air is accompanied by a characteristically loud noise. All the swallowed air may be expelled at one time or the expulsion may be gradual and brought about by a series of noisy belchings. The attacks of aërophagia may last for hours and even for days. There is a tendency for the disturbance to disappear and to reappear again if the individual is subjected to undue nervous strain.

Diagnosis and Treatment.—The diagnosis offers no difficulty. The most essential thing in the treatment is to convince the patient that the regurgitated air has nothing to do with fermentation, and to explain the mechanism by which aërophagia is brought about. As soon as the patient is thoroughly convinced that the symptoms are in no sense serious they usually can overcome them voluntarily. When a serious functional nervous disturbance underlies the condition, it obviously calls for treatment. Regulation of the diet, mastication, and regular meals are helpful. In some instances the use of bromids, chloral and nerve sedatives will promptly relieve the condition.

PNEUMATOSIS

Pneumatosis is characterized by over-distention of the stomach with air which cannot be expelled. The condition is said to be due to simultaneous spasm of the pylorus and cardia. Pneumatosis may occur in a pure neurosis associated with some general functional nervous disturbance. On the other hand, it is observed in association with organic lesions of the stomach, notably atony and dilatation. It is more frequently encountered in men than in women.

Symptomatology.—Two forms of the condition have been described, an acute and a more chronic type. In the acute form sudden over-distention of the stomach occurs giving rise to a ballon-shaped tympanitic tumor in the abdomen, which interferes with the diaphragmatic movements, and is often associated with dyspnea, cyanosis and palpitation, arhythmia and even collapse. The more chronic form usually follows the ingestion of a meal or over-exertion. The distention of the stomach is less acute and although dyspnea, cyanosis and palpitation occur in conjunction with marked epigastric discomfort, the symptoms are not so alarming as in the acute form. The patient is unable to spontaneously relieve himself by belching, and unless there is some outside interference the symptoms do not pass off until the spasm of the cardiac and pyloric muscles relaxes. In this condition there is nothing significant about the gastric analysis.

Treatment.—The acute forms require prompt treatment, and relief is best afforded by the passage of a **stomach tube.** In the management of all forms of pneumatosis, attention must be paid to the state of the nervous system. Various forms of mechano- and hydrotherapy are frequently found useful. Numerous drugs have been employed. Those in which chief reliance can be placed are the **bromids, eserin** and **strychnia.** The use of morphia is advocated by some.

Nervous Vomiting

The mechanism by which vomiting occurs is a complicated one. The act is under the control of the vomiting center located in the medulla in close association with the ganglia of the tenth nerve. To this center afferent fibers come from diverse portions of the body. The most important of these afferent fibers arise in the mucous membrane of the fauces and pharynx as well as from the mucous membrane of the stomach and the duodenum. Other afferent sensory fibers take their origin from various abdominal viscera. Anything which causes irritation of the sensory fibers or which irritates the vomiting center directly, may cause vomiting. It is therefore not surprising that vomiting occurs as a symptom of many conditions. It often accompanies disease of organs remote from the stomach. When the vomiting center becomes stimulated, the cardiac end of the stomach becomes perfectly relaxed and flaccid. Strong peristaltic waves keep the right half of the stomach and pylorus tightly contracted, whereas the left half of the stomach is entirely relaxed. While the stomach is in this condition, forcible contraction of the diaphragm and abdominal muscles expels the contents of the relaxed cardiac end of the stomach into the esophagus, where waves of antiperistalsis complete its expulsion.

We are here not concerned with vomiting which is due to nervous disturbances. The amesis which results from the ingestion of putrefactive or irritating food and the vomiting which is due to organic disease of the stomach and other organs may be dismissed.

Types of Nervous Vomiting.—Various subdivisions of nervous vomiting have been described. The simplest grouping and that which is generally employed, is as follows: Cerebrospinal vomiting, reflex vomiting, and nervous vomiting proper.

Cerebrospinal Vomiting.—This is due to central stimulation of the vomiting center, and occurs in association with many organic nervous diseases, which involve the central nervous system, notably brain tumor, brain abscess, apoplexy, concussion, encephalitis and the various forms of inflammatory disease of the meninges. It also occurs in transverse myelitis, and in association with the gastric crises of tabes dorsalis. In cerebrospinal vomiting the emesis is but a symptom of the underlying organic nervous disease.

REFLEX VOMITING.—This is frequently encountered and results from afferent impulses that are carried to the vomiting center by disease of various organs, other than the stomach: for example, diseases of the intestines, gall-bladder, appendix and genito-urinary organs. It is frequently observed in pregnancy. Reflex vomiting may also be associated with disease of the middle ear, of the eyes and of the pharynx.

TRUE NERVOUS VOMITING.—This is the type which is associated with various functional disorders of the nervous system such as neurasthenia and hysteria. It is seen more often in women than in men. Several types of nervous vomiting proper have been described.

Hysterical Vomiting.—In this type, which is found largely in women, vomiting occurs repeatedly day after day; sometimes after each meal, sometimes only once a day. In spite of this persistent vomiting, the general condition of these patients suffers but little; even after they have continued to vomit for weeks their bodily vigor is surprisingly good. The only explanation of this fact is that a certain amount of each meal is retained and only a portion is vomited.

Periodical Vomiting.—Leyeden described a form of vomiting in which the paroxysms recur at regular intervals. Between the attacks the patient is in good health. Paroxysms of vomiting may be of short duration or may be prolonged for days. The onset is usually accompanied by epigastric pain. At first the vomitus consists of the food recently eaten. Later only bile and mucus are ejected, and if the attack continues long enough, distressing retching without the ejection of any gastric contents may occur. When these periodic attacks recur at short intervals and are of considerable duration, the general health of the individual ultimately suffers, and in some instances alarming degrees of emaciation result.

Juvenile Vomiting.—This is a form of nervous vomiting observed in neurotic children, usually of a school age, especially those who have a tendency to overstudy or whose nervous systems bear badly the discipline and strain of school life. Such children will have attacks of vomiting from time to time without any apparent cause. That these attacks bear some relation to the strain of study is suggested by the fact that they usually clear up promptly when the child is taken out of school and never occur during the vacation time.

Psychic Vomiting.—This may occur in healthy robust individuals, as the result of some sudden shock or fright. Examples of this form of vomiting are furnished by those individuals who vomit whenever they see distressing occurrences, such as an accident.

CYCLIC VOMITING.—Another type of vomiting which is sometimes included under nervous vomiting, is known as the cyclic, or periodic vomiting of children. It is questionable whether this type of vomiting really falls in the category of nervous vomiting. There is considerable evidence to show that these recurring attacks observed in children between the ages of two and ten, usually ushered in by malaise, headache, anorexia and abdominal distress, and nearly always accompanied by fever, are in reality toxic in origin. There are a number of observations that indicate that the toxemia is the result of some metabolic disorder, which results in the production of abnormal quantities of acetone, and diacetic acid, beta-oxybutyric acid, sometimes lactic acid and nearly always an increase in indican. All or any of these substances may be responsible for the toxemia. This type of cyclic vomiting that is found in children is here mentioned only to be excluded from the group of true nervous forms of vomiting.

Symptomatology.—All these forms of nervous vomiting proper have certain characteristics in common which aid materially in their recognition. As a rule vomiting is accomplished with great ease and without any retching and distress. The quantity of food taken bears no relationship to the vomiting. Often highly irritating substances may be ingested with impunity, whereas easily digested materials are promptly rejected. In some instances of nervous vomiting, only one form of food can be ingested. Another characteristic worthy of note is the insignificant effect which prolonged nervous vomiting has upon the general condition of the patient. In most cases of nervous vomiting, the associated symptoms point to the existence of some fundamental functional weakness of the central nervous system.

Diagnosis.—The diagnosis of nervous vomiting is by no means easy. It is important to determine the exact type of nervous vomiting with which one is dealing, this is often a most difficult matter. As in the diagnosis of other forms of gastric neurosis, it is essential to exclude all possible organic disease. The frequency with which vomiting occurs in many diseases of the stomach and other organs, makes the diagnosis of nervous vomiting both dangerous and difficult. A thorough clinical study of the patient, a complete investigation of the gastro-intestinal tract, and attention to the above enumerated characteristic features of nervous vomiting will aid materially in arriving at a proper diagnosis.

Treatment.—The first consideration in the treatment of nervous vomiting is to find and **remove the cause.** Since most instances of true nervous vomiting are but local manifestations of a general functional derangement of the central nervous system, the treatment appropriate to the nervous condition is urgently indicated. In some instances hygienic measures, change of scene, regulation of exercise, etc., will prove sufficient. In the more severe cases, a carefully conducted rest treatment is required. No definite rules can be laid down for the proper dietetic treatment of these cases. As a rule, liquids are better borne than other forms of food. In some instances, however, liquids are badly tolerated, and that form of diet must be found which the patient can retain the best, since the essential thing is to increase the patient's nutrition. When nothing can be retained, rectal alimentation may be resorted to. Glucose and sodium bicarbonate enterocylsis is often useful and in recent years the **duodenal tube** has been successfully employed, and perhaps offers the most effective means of managing the severe cases. An endless array of drugs have been tried. Morphia is probably the most effective, but should never be used except as a last resort, and then should only be given hypodermically and if possible the patient should not be aware of what is being administered. The well-known combination of a powder which contains a twelfth of a grain of **cocain hydrochlorate,** a twelfth of a grain of **codein sulphate,** and a grain of **cerium oxalate,** administered dry on the tongue every few hours, frequently proves beneficial.

Prognosis.—In nervous vomiting proper the prognosis is usually good, provided of course the underlying disturbance of the nervous system can be corrected. In the forms of vomiting described as reflex and cerebrospinal, the prognosis depends obviously upon the underlying cause, which is frequently serious and incapable of removal.

Nervous Nausea

Like vomiting, nausea is a frequent symptom of many diseases, and its mechanism is closely related to that of vomiting. Occasionally it is not due to any organic disease of the stomach or other organs, and is encountered as the accompaniment of neurasthenia, hysteria or functional derangements of the central nervous system; under such conditions it may be looked upon as a form of true gastric neurosis. Nervous nausea is more common in women than in men; is frequently observed in chlorotic young women; and may prove a troublesome symptom at the menstrual period and at the menopause. Like nervous vomiting, nervous nausea may bear no relationship whatsoever to the ingestion of food. It may occur at any time during the day or night, it may be intermittent or continuous. Its duration is variable and may lead on to true nervous vomiting. The secretory and motor functions of the stomach are unaltered in most cases of nervous nausea. The diagnosis can only be made when all possible organic disease has been excluded. The management of nervous nausea does not differ essentially from the treatment outlined for nervous vomiting.

Cardiospasm

By cardiospasm is meant a spasmodic contraction of the cardia. Cardiospasm occurs in the course of numerous organic conditions, involving the lower end of the esophagus and the cardiac end of the stomach, such as ulcers, fissures, neoplasms and inflammatory lesions. When any such anatomical basis exists, the cardiospasm cannot be regarded as a neurosis. On the other hand, cardiospasm rather frequently accompanies neurasthenic and hysterical states without any organic basis. Under such conditions it is justifiable to regard it as one of the forms of gastric neurosis. Cardiospasm is frequently associated with weakness or relaxation of the esophageal wall, and when it is persistent or recurrent, sooner or later may bring about actual dilatation of the esophagus. The exact mechanism of cardiospasm is not clear. Most writers seem willing to accept the suggestion of Meltzer as a likely explanation, namely, that cardiospasm is the result of a failure of the normal inhibitory influences of the central nervous system which control the contractions of the cardiac sphincter.

Etiology.—Cardiospasm is more common in women than in men. As in the case in most gastric neuroses, it is more common between the twentieth and fortieth years. When functional in its origin, it is associated with various neurasthenic states, and is frequently brought on by excitement, worry, depression and fear. Too rapid eating, food that has been imperfectly chewed, highly seasoned and stimulating food, the ingestion of food that is too hot or cold may frequently precipitate the attacks.

Symptomatology,—There are two varieties of cardiospasm, the acute and chronic. In the acute form the patient is well between the attacks which come on periodically, and last but a day or two. At the onset of the attack, the patient experiences painful and difficult deglutition, a feeling of fullness and oppression, and a burning sensation is frequently felt behind the sternum. As food is ingested it accumulates in the esophagus in spite of violent efforts to force it into the stomach. Finally regurgitation of undigested alkaline food takes place and with evacuation of the contents of the esophagus, the symptoms disappear until the next acute recurrence.

In the more chronic form of cardiospasm, the patients suffer more or less constantly from dysphagia. For short periods they are able to take their regular meals with a fair amount of comfort. But the dysphagia gradually returns, and varies in degree from time to time. As a rule semi-solids and liquids may be swallowed with some degree of ease. Frequently solid food cannot be forced past the cardia, except when it is thoroughly chewed and taken in small quantities along with the liquids. Fullness, pressure and discomfort are frequently complained of. As a chronic cardiospasm persists, sooner or later diffuse dilatation of the esophagus results. The greater the degree of esophageal dilation, the more substernal discomfort is experienced. Violent efforts are constantly being made to force the contents of the esophagus past the spastic cardia. More or less intermittent regurgitation of the undigested alkaline contents of the esophagus takes place. When chronic cardiospasm becomes persistent, the general nutrition invariably suffers.

Diagnosis.—The diagnosis of functional spasm of the cardia must be made from cardiospasm associated with organic disease. It is essential to differentiate cardiospasm from esophageal obstruction, the result of neoplasms at the lower end of the esophagus and cardia, and from external compression on the esophagus from tumors, aneurysms, mediastinal abscesses, enlarged mediastinal glands, effusions and adhesions. To-day by far the most satisfactory way of recognizing cardiospasm and eliminating some of the possible organic causes for cardiac obstruction is afforded by the x-ray. In all cases of cardiospasm, the patient should be subjected to careful roentgenological examination. Examination by means of the esophagoscope affords another important diagnostic method. In addition to these procedures, passage of the esophageal bougie, furnishes valuable information. In cardiospasm of functional origin less resistance is offered to esophageal bougies of large caliber than to small ones. When a large caliber esophageal bougie is passed to the point of constriction, a sense of resistance will be felt at first which suddenly gives way allowing the bougie to pass into the stomach. The contrary is the case when an organic basis exists for cardiac obstruction.

Treatment.—In all forms of cardiospasm of nervous origin, particular care should be taken to remove if possible the underlying nervous cause, to improve the general nervous condition and general nutrition of the patient. In the acute form of this condition, the cardiospasm may usually be terminated by the introduction of a large-sized **esophageal bougie** into the stomach. When once introduced it should be allowed to remain for some time. Various drugs having a tendency to relax the spasm of the cardiac muscle have been found useful. Of these **belladonna** is undoubtedly the most efficient. The bromides and chloral are sometimes helpful and in extreme cases morphia has to be resorted to.

The treatment of the chronic form offers more difficulty. In addition to doing everything possible to bring the general nervous tone back to normal careful feeding should be practiced. Frequently only semisolid or liquid food can be taken. In the extreme cases feeding has to be carried out through a tube introduced through the cardia. Repeated dilatations of the cardia by large-sized bougies allowed to remain in position for some time, should be practiced; they are sometimes sufficient to overcome the chronic spasm. The most effective local measure, however, is forcible dilatation by means of some of the well-known dilators that are now available.

PYLOROSPASM

The term pylorospasm refers to spasmodic contractions of the pyloric sphincter.

Etiology.—Spasm of the pylorus is brought about either by local irritation of the mucosa of the pylorus, as occurs in erosions, fissures, ulcers, neoplasms involving the pylorus, or by conditions associated with gastric hyperacidity, or by reflex irritations of the pylorus due to inflammatory disease of organs elsewhere in the abdomen such as is observed in disturbances of the duodenum, diseases of the biliary tract, the pancreas and the appendix. In none of the above-mentioned conditions can the spasm of the pylorus be looked upon as functional in character. There are, however instances of pyloric spasm for which there is no discoverable organic cause, and which appear to be characteristic of some functional nervous disturbance. It is only in this latter group that pylorospasm can be looked upon as a gastric neurosis, and although pylorospasm is a condition frequently encountered, it is undeniably seen with comparative rarity as a true neurosis. In all probability pylorospasm of functional origin is the result of excessive vagal stimulation and may be looked upon as one of the manifestations of vagatonia.

Symptomatology.—In the beginning pylorospasm of nervous origin occurs at irregular intervals, and lasts but a short time. Gradually the attacks become more frequent, and the duration of the spasm increases. As a rule the spasmodic contraction takes place two or three hours after the ingestion of a meal at the hight of digestion. This contraction is associated with varying degrees of cramp-like pain in the region of the pylorus. With marked grades of the affection in individuals with thin abdominal walls, a mass due to the firmly contracted pylorus may at times be felt. At first there is no noteworthy retention, but as the pylorospasm becomes more persistent and spasticcontraction of the pylorous exists more or less continuously, there gradually takes place dilatation of the stomach, with associated retention. When the condition reaches this degree, periodic attacks of vomiting and retained gastric contents occur. With the vomiting there is prompt relief of the pain and the other symptoms. The gastric contents as a rule show an increase in acidity.

Diagnosis.—Before hazarding a diagnosis of plyorospasm due to nervous origin, it is essential that all the many organic causes for plyorospasm, both within and without the stomach, be excluded. The existence of a pylorospasm, and the retention subsequent thereto, is easily recognized. To determine the underlying cause of the pyloric cramp is often a difficult matter. The diagnosis is materially helped by roentgenological examination of the gastro-intestinal tract. In the hands of a competent roentgenologist, a purely functional pylorospasm can usually be differentiated from one due to organic disease. Friedenwald and others emphasize the fact that the administration of large doses of atropin furnishes a useful means of differentiating between nervous and reflex pylorospasm and that due to organic disease. In the former instances, atropin will relieve the spasm, but in organic disease of the pylorus, the spasm is unaffected by atropin.

Treatment.—When pyloric spasm is nervous in origin, the underlying functional disturbance of the nervous system must be eliminated if possible. When the pylorospasm is reflex in origin, the cause of the condition must be found and removed.

Inasmuch as in nearly all instances of pylorospasm, gastric hyperacidity exists which tends to keep up the condition, regulation of the diet, the exhibition of those drugs which tend to control the hyperacidity, such as have been discussed under the treatment of gastric ulcer and hyperchlorhydria, often afford considerable relief. The drug which tends to relieve the spasm of the pylorus better than any other, is **atropin** given hypodermically in large doses. Opiates, in the form of morphia or some of its derivatives, may be required in extreme cases of pylorospasm. When retention and dilatation occur, **gastric lavage** is a most useful procedure. In some severe cases of spasm of nervous origin, **pyloroplasty** has been practiced with brillant results.

ATONY OF THE STOMACH

There is a question in the minds of some observers, as to whether atony of the stomach can ever be looked upon as a gastric neurosis; whether it is not always but a part of gastric dilatation. It is probably true that in a majority of cases atony of the stomach cannot be classed as one of the gastric neuroses. Nevertheless, there are instances of gastric atony which it seems justifiable to include under functional gastric disorders.

By atony of the stomach is meant a condition in which the normal tonus of the gastric musculature is lost. When this occurs, it delays the evacuation of the gastric contents into the intestines. If this condition of affairs persists for any length of time, gastric dilatation ultimately supervenes.

When one recalls that the rhythmic contraction of the smooth muscle of the stomach is dependent upon the maintenance of normal muscle tonus and that this tonus, in part at least, is dependent upon a normal equilibrium between the impulses that are conveyed to the gastric musculature through its extrinsic nerves, the vagi and the sympathetics, it is conceivable that disturbances of the nervous system which modify the discharge of normal impulses along these extrinsic nerves, may through vagal inhibition or sympathetic stimulation, so modify the normal tonus of the gastric wall as to bring about atony. Under such conditions, and when there is an absence of any organic lesion of the stomach, the atony may be looked upon as nervous in origin.

Etiology.—In individuals who suffer from that disturbance of the automatic nervous system referred to by Eppinger and Hess as sympatheticotonia, psychic disturbances and prolonged anxiety may produce atony. In individuals of this type the ingestion of large quantities of food or fluid, or the prolonged use of irritating foods, may induce the condition.

Symptomatology.—In primary atony of this type, especially in its early stages, dilatation of the stomach does not occur. The symptoms are chiefly due to motor insufficiency, but the gastric fullness, discomfort and distention after even small meals, is characteristic. The appetite is diminished, and the symptoms gradually subside as the stomach becomes empty, only to recur on the taking of food. Sour eructations and belching are common symptoms. Nausea and vomiting are not characteristic of the condition.

In the early stages of atony, physical examination may show nothing, but after dilatation takes place, the stomach is found to be enlarged. After the ingestion of even small quantities of fluid, the characteristic splash can be heard over the gastric area. Examination of the stomach contents reveals a certain amount of retention, but as a rule the acidity is little if any altered from the normal. In most cases of atony, constipation is the rule.

Treatment.—In view of the impaired motor power of the stomach, and the tendency to dilatation, it is obviously essential that the viscus must not be overloaded and that the food which enters it should be as well prepared as possible. For that reason those who suffer from atony of the stomach, should be instructed to eat slowly, to masticate thoroughly, and to take small quantities of food at frequent but regular intervals. These meals should consist of food that is easily digestible and non-irritating. Bulky foods, and large quantities of fluid should be avoided. Every effort should be made to tone up the musculature of the stomach and the abdominal walls. This may be helped by **systematic exercises, massage** and **hydrotherapy**. Any associated constipation should be overcome. The general condition of the patient should require special attention, and all associated nervous disturbances should receive the appropriate treatment. In many cases considerable benefit is derived from placing the patient upon ascending doses of the tincture of **nux vomica.** In an ordinary adult, 15 drops may be given before meals, and the dosage increased by 2 drops per dose every third day, until as much as 30 drops are being taken three times a day. Patients may be kept on this dose from several days to a week, if it is well borne; then the dose should be reduced in the same way that it was increased.

DISTURBANCES OF SECRETION

HYPERCHLORHYDRIA

Of all the secretory disturbances of the stomach, that which is characterized by an increase in the free hydrochloric acid during the period of digestion, is by far the most common. Lockwood, summarizing the experience of a number of observers, as to the frequency of hyperchlorhydria, states that Jaworski encountered this secretory abnormality in 75 per cent. of all cases that had indigestion. Einhorn found it in 50 per cent. of cases; Friedenwald in 63 per cent.; in 2,000 private patients, Fenwick observed hyperacidity in 9.2 per cent., and in 4.8 per cent. of his hospital cases. Lockwood himself found simple hyperacidity in 13.8 per cent. of his patients.

Etiology.—Hyperchlorhydria is frequently found associated with organic disease of the stomach, notably in gastric ulcer and in the acid form of chronic gastritis. It also occurs with great frequency as the result of reflex irritation from diseased conditions of the abdomen outside of the stomach. This is particularly true of chronic appendicitis, chronic cholecystitis and peritoneal adhesions. Hyperchlorhydria has also been found associated with the gastric crises of tabes dorsalis. In spite of the fact that hyperchlorhydria is so frequently encountered in connection with organic disease, nevertheless a considerable group of cases exist in which no organic cause for the condition can be found and which may be looked upon as true gastric neuroses. It is with this type of hyperchlorhydria that we are here concerned.

Considerable discussion has arisen as to just what degree of gastric acidity is necessary before the condition can be looked upon as one of hyperchlorhydria. This difficulty is increased by the fact that there is no unanimity of opinion as to just what constitutes normal gastric acidity. At one time when sole reliance was placed upon the single tube test, the normal acidity of the stomach following a standard test such as the Ewald breakfast, was looked upon as fairly fixed, and 50 or over, reckoned in terms of decinormal sodium hydroxid solution (0.2 per cent. HCl), was looked upon as the upper limit of normal. Recently, however, much light has been thrown upon the subject by the work of Rehfuss and his associates, who adopted the fractional method of gastric analysis whereby the gastric acidity could be observed throughout the entire period of digestion. Rehfuss and others have clearly shown that hyperacidity may occur at any time during the digestive period. The older method of removing the test meal one hour after its ingestion frequently gave erroneous results, since at that time the acidity may be normal, although before or after that time there may be marked departures from the normal values. The work of Rehfuss also suggests that even in normal people there is individual variation. What may be the normal acidity in one person, may be entirely abnormal for another. If we accept this conception of normal individual variation of the gastric acidity, it becomes impossible to say dogmatically, what degree of acidity can be regarded as pathologic. When the degree of acidity in the stomach of an individual reaches a point in excess of the acidity that is normal for that individual, symptoms are likely to result and hyperchlorhydria may be said to exist. This theory also explains the well-known clinical fact that high degrees of acidity show no symptoms in certain individuals, whereas much lower degrees of acidity, or even values below normal, produce marked symptoms in others. On the other hand, it is held by some that this variability in the subjective symptoms produced by gastric acidity depends more upon the sensitiveness of the mucous membrane than upon the actual degree of acidity. In other words, that hyperesthesia of the stomach is a necessary factor in bringing about the symptoms of hyperchlorhydria.

Hyperchlorhydria of pure nervous origin, is more often observed in young people than in the old. It is more often found in those who lead confined lives, who live under a tension, and work with their brain, than in those whose occupations keep them out of doors and employed at physical labor. Undoubtedly dietetic indiscretions, such as the constant eating of rich, highly seasoned and irritating food, the ingestion of large quantities of cold drink, the excessive use of alcohol, in some individuals, the excessive use of coffee and tobacco, all may lead to hyperchlorhydria.

Hyperchlorhydria is more common among the well-to-do than among the poorer classes. It is also observed with great frequency among those races that are characteristically neurotic, notably among the Hebrews. Gastro-intestinal atony, especially when associated with colonic stasis, frequently is the cause of hyperchlorhydria.

Symptomatology.—The symptoms of hyperchlorhydria are, as a rule, characteristic. Their onset is rarely acute, the condition developing gradually. The most conspicuous symptoms are eructations of acid material (pyrosis), heart-burn, burning in the stomach and epigastric pain. Occasionally the pain comes on soon after eating, but as a rule it is more marked from two to three hours after taking food. When the pain is at its height, eructations of sour material occur and afford a certain amount of relief. The heart-burn is the result of irritation of the esophagus by the acid material that is eructated. The pain of hyperchlorhydria varies from epigastric discomfort to severe gastralgia. The severe pain is felt in the epigastrium and at times it is referred to the back between the shoulder blades. It is not associated with local rigidity, and pressure on the epigastrium affords relief rather than aggravating it. The severity of the symptoms varies considerably with the type and quantity of food ingested. Irritating acid foods increase the acid erucations and make the pain worse. The pain is induced more by starchy food than by protein. The symptoms come on more quickly after small meals than after large. The characteristic of the pain of hyperchlorhydria is that it is nearly always relieved by the ingestion of food or even water and the taking of alkalies or by vomiting. It is unusual for the pain to persist during the night.

The general nutrition of patients with hyperchlorhydria is rarely much impaired. The appetite is good, sometimes it is excessive. Increase in thirst has been noted. Vomiting is rare, constipation is the rule. Physical examination is negative, there being an absence of local tenderness or rigidity.

In uncomplicated hyperchlorhydria examination of the gastric contents does not show any disturbances of motility; retention does not occur and hypersecretion is absent. Gastric analysis shows characteristically a marked increase in the free hydrochloric acid content of the stomach, starch digestion is poor, and protein digestion is correspondingly increased.

Diagnosis.—In view of the frequency with which hyperchlorhydria occurs as a symptom associated with various organic diseases, the importance of careful diagnosis is obvious. Hyperchlorhydria must be differentiated from ulcer, both gastric and duodenal. The regularity with which hyperchlorhydria accompanies gastric ulcer, makes this diagnosis exceedingly important and very difficult at times. As a rule the symptoms are more severe and constant in ulcer and there are more definite circumscribed areas of epigastric pain and tenderness. The more frequent occurrence of occult blood, the history so characteristic of ulcer, the symptoms extending over a long period of time, and in duodenal ulcer particularly, marked remissions occurring in the pain, are all helpful diagnostic points. When these are taken in conjunction with careful roentgenological examination of the gastro-intestinal tract the differential diagnosis can usually be made. Other conditions from which hyperchlorhydria must be differentiated are acid gastritis, chronic hypersecretion, atony and dilatation of the stomach and gall-stones.

Treatment.—The chief indications as to treatment are to remove any underlying cause and to prevent irritation of the stomach. The latter requirement is largely accomplished by careful **regulation of the diet**, which is perhaps the most essential thing in the treatment of hyperchlorhydria. It is necessary to exclude from the diet all acids, all condiments, spices and highly seasoned dishes of all kinds. Fried food should be prohibited. All stimulants, such as alcoholic drinks, tea, coffee as well as hard foods that irritate mechanically, must be eliminated. The diet should be a bland, mixed, nutritious and easily digested one. It is a distinct advantage to have the diet liberal and to have it contain an adequate amount of proteins, carbohydrates and fats. All fresh bread, hot bread, hot cakes, etc., should be prohibited. Such breads as are taken should be in the form of dried, toasted, pulled or stale bread. In severe cases it is of advantage to have foods finely divided, the vegetables put through a colander, the cereals strained and meats chopped fine. Proteins are best given in the form of chicken, squab, fish, and eggs, soft-boiled or poached. Fats in the form of butter, olive oil, and cream, are particularly useful, and tend to lessen the acidity. The meals should be regular and should be eaten slowly and away from all disturbing nervous influences. Patients should be instructed to masticate thoroughly. In many instances it is an advantage to have the patient take between-meal feedings, or have the patient take five small meals a day, rather than three large ones. Moderate water drinking is permissible. Tobacco, if used to excess should be prohibited.

In addition to the dietetic regulations, much may be done by regulating the personal hygiene of individuals. If the patient is not too much below par generally, it is of advantage to have them take **light setting-up exercises** every morning, followed by a cool sponge or tub bath and a rub-down. A short walk in the open air before meals is of use, and when it can be done, out-door exercise such as golf, riding and walking are of great value. When hyperchlorhydria occurs in tired, overworked individuals, a vacation in the country or at the seashore, or the relief from routine work afforded by a journey or a camping or fishing trip has often proved of the greatest benefit. In emaciated, anemic and run-down individuals, nothing is so effective as a well carried out **rest treatment.** The importance of building up the general condition of the patient and restoring the tone of the tired nervous system, must not be overlooked. It is often quite as essential as the avoidance of local gastric irritation.

In hyperchlorhydria, drugs properly administered are of great benefit. Alkalies in the form of combinations of **bicarbonate of soda**, the **heavy oxid of magnesia** and the **subcarbonate of bismuth**, administered in powder form, at the height of digestion, that is about two hours after meals, often afford considerable relief. The best results are obtained by giving the alkalies in adequate doses. The oxid of magnesia has a laxative effect and helps to overcome the constipation. In order to reduce the hyperesthesia of the stomach, and to lessen secretion, combinations of **silver nitrate** in doses of from 1/6 to 1/4 grain (.0108 to .0162 gram), and the **extract of hyascyamus** or **belladonna** in doses of 1/12 grain (.0054 gram), taken half an hour before meals, are most effective. In certain nervous individuals nerve sedatives such as combinations of **bromids**, **luminal** or even small doses of **chloral** are serviceable. It should be emphasized, however, that without proper attention being given to the dietetic and hygienic management of these cases, little can be expected with drug treatment alone.

Prognosis.—The prognosis in uncomplicated hyperchlorhydria is good. The shorter the duration of the case, the better is the prognosis. It is rarely possible to permanently reduce the hyperacidity, but it is possible to relieve the symptoms very markedly. Even when cases have been apparently relieved of symptoms for some time, recurrences are liable to occur.

ACHYLIA GASTRICA

By achylia gastrica is meant a total absence of gastric secretion, both free hydrochloric acid and the ferments being lacking. By far the greatest number of cases of achylia gastrica are associated with severe organic lesions of the stomach, or are part of the clinical picture of pernicious anemia. There is, however, a group of cases in which achylia gastrica can be demonstrated but for which there is no discoverable organic basis. It seems justifiable, therefore, to classify this group among the gastric neuroses.

Etiology.—The etiology of simple achylia gastrica of functional origin is not clear. Some cases apparently develop in early youth and may be congenital. In others the digestion, or lack of gastric secretion, seems to be an accompaniment of a general underlying nervous disturbance. According to Stockton, many cases of achylia gastrica, in the beginning at least, are the result of depression in the functional activity of the gastric secretory glands. The condition has been encountered with equal frequency in both men and women. It is rare to encounter it in early life.

Symptomatology.—A small number of the cases of achylia gastrica of nervous origin, go through life without any gastric or intestinal symptoms, and without impairment of their general health. In the majority of the cases of achylia gastrica, however, various gastric symptoms are conspicuous. These symptoms include loss and variation of the appetite; fullness and discomfort in the epigastrium; sometimes pain, which is rarely severe and occurs on the ingestion of food and persists for a variable length of time. Epigastric burning, eructations of sour material, and belching of gas sometimes occur. Vomiting is rare. In another rather definite group of achylia gastrica cases, there are no gastric symptoms, but intestinal symptoms dominate, the most conspicuous of which is persistent troublesome diarrhea. In view of the variability of the symptoms, the condition can be recognized only by careful gastric analysis. A single tube test is not satisfactory, since at the end of the first hour a case of delayed hyperacidity may strongly simulate an achylia. In order to justify the diagnosis, several specimens of gastric juice removed during the digestive period should show a complete absence of hydrochloric acid and gastric ferment. The total acidity in achylia gastrica varies between 2 and 10 in terms of decinormal sodium hydrate.

Diagnosis.—The diagnosis has to be made from carcinoma and from chronic gastritis. Repeated gastric analysis, examinations of the stools and careful x-ray studies are the most useful aids in such diagnosis.

Treatment.—As in other forms of secretory disturbances of the stomach, so in achylia gastrica, the most essential feature of the treatment is careful **dietetic regulation.** Both the quality and quantity of the diet require **careful adjustment.** Since the ability of the stomach to properly prepare food for the intestines is markedly diminished, it is essential that the diet should be of such a character that in spite of the digestive inadequacy of the stomach, it enters the intestines as nearly prepared as possible for further digestion. For that reason non-irritating, easily digested foods are preferable, and it is best for all foods to be taken in a finely divided state. In severe cases it may be necessary to restrict the diet to liquids, cereals and vegetables which should be put through a colander, and all irritating cellulose particles removed. Meats, such as the white meat of chicken, chops, sweetbreads and fish should be taken in small amounts, and always in a finely divided form. Soups are useful and wherever possible vegetables should be given as purées. All vegetables, and certainly starchy ones such as potatoes, should be well cooked and the latter should be taken only as mashed potatoes. Boiled eggs are allowable. In most instances milk is well borne. Occasionally individuals are encountered who have an idiosyncracy toward milk and are unable to take it. Fats should be given sparingly, since they have a tendency to further inhibit gastric digestion.

The quality of food ingested at one time is another important consideration. Since it is essential to conserve the motor power of the stomach in this condition, large quantities of food are unwise. Frequent small feedings in most instances are better borne. Owing to the gastric discomfort following meals, and the lack of appetite frequently encountered in achylia, many patients voluntarily cut down their diets so that the amount of nourishment they are getting is entirely inadequate. This tendency must be guarded against, and although great care should be exercised in the regulation of the diet, it is of equal importance that patients receive a sufficient number of calories to maintain their nutrition.

General measures directed towards improving the underlying neurasthenic states and toning up the general musculature, should not be neglected. Moderate exercises out doors, massage, hydrotherapy, etc., are indicated in those whose general condition permits of such procedures. In those who are debilitated and below par, rest is essential.

As a rule drugs are of little value. Some cases do well on ascending doses of the tincture of **nux vomica** before meals, given in the manner described under Atony of the Stomach. A great many cases are markedly improved symptomatically by the administration of from 15 to 20 minims of **dilute hydrochloric acid** in three-quarters of a glass of water taken slowly through a tube with meals.

Prognosis.—In simple achylia gastrica the prognosis for reasonably good health is good. The condition is probably never cured, but so long as gastric dilatation does not occur, patients can go through their entire lives and suffer little discomfort.

GASTROMYXORRHEA

By gastromyxorrhea is meant a condition in which there is an a normal flow of mucus, either intermittent or constant from the gast glands. In a normal fasting stomach quantities of mucus may be found from time to time. In order to regard the condition as one of gastromyxorrhea, according to Kuttner and others, at least 25 c.c. of mucus must be found in the fasting stomach. Two forms of the condition are described, the intermittent form which recurs at varying intervals, and the continuous form.

Intermittent Gastromyxorrhea.—The intermittent form is rarely encountered. The exact cause of the condition is not understood. The attacks come on at irregular intervals, and usually have their onset in the early morning. The most obtrusive symptom is persistent and intractible vomiting. The vomitus contains large quantities of mucus. During the attack absolutely nothing can be retained on the stomach. The attack may be preceded by malaise, headache, and nausea. The vomiting may last for several hours or for several days. In the cases in which the vomiting continues for some time, patients become exhausted, and dehydrated. The abdomen is retracted, the urine is diminished. Treatment is of little avail, and the attacks end spontaneously, when the vomiting disappears, and the patient is again able to take food. For a variable time between attacks, patients enjoy good health and are free of gastric symptoms.

Continuous Gastromyxorrhea.—The continuous form of gastromyxorrhea is the more usual variety. In the majority of instances, this is associated with some organic disease, particularly gastric ulcer and chronic gastritis. In a few cases, however, the condition seems to be a true gastric neurosis and no associated organic condition can be demonstrated. There are no characteristic symptoms of the continuous form, the acute manifestations that accompany the intermittent variety are absent. The chief symptom is finding an abnormal amount of mucus constantly in the stomach upon gastric analysis. Without the aid of gastric analyses, the condition can rarely be recognized.

Diagnosis.—Whenever any form of gastromyxorrhea is suspected, the first thought should be the probable presence of some organic lesion in connection with it. In making the diagnosis care should be taken not to confuse mucus in the stomach derived from other regions such as the nose and throat, with that secreted by the stomach as occurs in this affection.

Treatment.—When the condition requires treatment, the first thing is to eliminate any possible underlying cause and institute measures designed to bring the patient's general condition back to normal. The associated gastric symptoms of the condition should be treated. In the acute form prompt lavage is one of the most effective methods of relieving the attack. In the continuous type of gastromyxorrhea, repeated **lavage** should be performed in order to rid the stomach so far as possible of the constantly accumulating mucus.

SENSORY DISTURBANCES REFERABLE TO THE STOMACH

A group of conditions characterized by pain and other sensations referable to the stomach, but unassociated with anatomical lesions of that organ, have been regarded as sensory gastric neuroses. Although for clinical conveniences this class of disorders may be designated as sensory, as a matter of fact the underlying cause of the abnormal sensation is increased tonus or spasm of the gastric musculature, and in accordance with the theory of viscerosensory reflexes above referred to, the abnormal stimuli thus produced are translated into pain and other sensations that have their seat in the skin and muscles of the abdominal wall. It will be recalled that the mucous membrane of the stomach is insensible to pain stimuli.

Gastralgia.—Properly speaking this term refers to severe paroxysmal gastric pain not due to organic causes. In recent years it has become more and more apparent, that pain of this character as a pure neurosis, is excessively rare. Careful analysis shows that all cases of gastralgia have an underlying organic basis, such as gastric or duodenal ulcer, carcinoma, various types of gastritis, gall-stones, gastric or duodenal adhesions, and pylorospasm secondary to disease of other abdominal organs, such as the appendix, as well as the visceral crises of tabes dorsalis, and lead poisoning. It is conceivable that occasionally under unusual conditions severe pain of functional origin may occur, but as a neurosis gastralgia would appear to be extremely rare. The diagnosis is certainly such a dangerous one that it should never be made or only when every possible organic cause of gastric pain can be definitely excluded.

Hyperesthesia Gastrica.—This is a term applied to undue sensitiveness of the gastric mucous membrane to all varities of stimuli, even normal ones. The condition is usually found in association with various forms of organic lesions, and it is said to explain why some patients with normal or subnormal gastric acidity have marked subjective symptoms, whereas others with marked hyperchlohydria are free of discomfort. As a pure neurosis it may be found in individuals with unstable and hypersensitive nervous systems.

Disturbances of Appetite.—Various perversions and alterations in appetite may be classed under the sensory neuroses. The term *bulimia* refers to an abnormal increase in the sensation of hunger. Opposed to this is a condition known as *anorexia nervosa,* which implies an entire absence or marked diminution of the sensation of hunger. *Paroxexia* is a term used to denote perversion of the appetite. In this condition patients manifest a desire for unusual and bizarre substances, such as earth, clay, chalk, unwholesome and unusual forms of food and even for disgusting substances. *Akoria* is a condition in which there is an entire lack of the sensation of satiety.

All these perversions and alterations of appetite may accompany various organic diseases of the stomach. They all, however, may result from a pure neurosis, in which case they are more common in women than in men, and are frequently associated with pregnancy, menstruation, hysteria and anemia.

Nervous Dyspepsia, or Neurasthenia Gastrica.—In this condition, although sensory disturbances are the most obtrusive, the symptoms of any or all of the various functional disturbances of the stomach may be present. The disorder is characteristically polysymptomatic and unlike most gastric neuroses, no single symptom stands out with undue prominence.

ETIOLOGY.—The condition is most frequently encountered in individuals of nervous temperament, and those who have been subjected to over-work, anxiety, and nervous strain. This syndrome also occurs as the result of dietetic errors, the excessive use of alcohol and tobacco and it may occur in hyperthyroidism, in secondary anemias particularly those of the chlorotic type, and reflexly as the result of disease of organs other than the stomach. Although it may occur as a pure neurosis, the syndrome is not infrequently associated with various organic diseases of the stomach and with many general diseases.

SYMPTOMATOLOGY.—The symptoms are essentially variable. Various forms of epigastric distress, fullness and discomfort amounting at times to actual pain are usually present. The appetite may be normal, diminished, or perverted. Vomiting is rare, eructations are common. A varied array of nervous symptoms are associated with those referable to the stomach. The general nutrition of these patients is usually good, physical examination so far as the stomach is concerned is negative. Gastric analysis shows no noteworthy departure from the normal, and there is no alteration in the motor power of the stomach. The subjective symptoms are most marked during the period of digestion, and their intensity is out of all proportion to any objective findings.

TREATMENT.—Treatment directed toward the stomach alone usually accomplishes but little. It is essential in cases of nervous dyspepsia to direct attention to the underlying condition of the nervous system. In those who are anemic, under weight and debilitated, **rest cures** bring about prompt relief. In those who are well nourished and reasonably robust but who are mentally and nervously depressed, **fresh air, exercise, hydrotherapy, changes of scene and relief from work and anxiety,** are the proper procedures. If any abnormal alterations in the secretions of the stomach exist, they should be corrected. Patients with this disturbance have usually voluntarily restricted their diet, and considerable difficulty is sometimes encountered in getting them to take an adequate, easily digested, mixed diet. So far as drugs are concerned, the best results are usually obtained from the use of the so-called tonics, of which **nux vomica** acts the best; when anemia is present, **iron** and **arsenic** are indicated.

PROGNOSIS.—The prognosis is usually good, if the underlying disturbances of the nervous system can be removed or improved.

Gastrosuccorrhea or Hypersecretion.—This disorder is characterized by the continuous excessive outpouring of gastric juice, and was originally described by Reichmann of Warsaw in 1882. The disorder is frequently referred to as Reichmann's Disease. At the outset it should be clearly stated that hypersecretion is an entirely separate condition from hyperacidity although it is true that the two states are frequently found in association.

Three forms of hypersecretion are generally described. First, continuous hypersecretion with periodic outbreaks of acute manifestations *(gastrosuccorrhea continua periodica)*; sometimes this form is designated as acute hypersecretion; this term, however, is misleading, since in reality the acute outbreaks are but exacerbations of a preëxisting chronic condition. The second variety is referred to as chronic hypersecretion, or *gasttrosuccorrhea continua chronica;* in both the above forms of hypersecretions, an excessive amount of gastric juice exists in the stomach, both during the digestive, interdigestive and fasting periods. A third variety, which is known as digestive or alimentary hypersecretion, is characterized by the presence of an excessive flow of gastric juice only during the digestive period.

The fasting contents of the normal stomach do not amount to over 10 or 20 c.c. Any quantity in excess of these figures must be looked upon as evidence of hypersecretion. In a series of cases studied by Crohn and his colleagues, by means of the fractional method, the total amount of gastric contents withdrawn varied from 10 to 325 c.c. The average amount which was above normal and represented hypersecretion was 102 c.c. The same authors found that in 20 per cent. of their series of gastric cases, hypersecretion was present. As has before been mentioned, in a majority of cases of hypersecretion some degree of hyperacidity exists. In Crohn's series the average free hydrochloric acid content in the cases of hypersecretion amounted to 35, the average total acidity was 64.3.

ETIOLOGY.—It was first thought that hypersecretion was chiefly a functional secretory disturbance. Pathological, roentgenological and surgical observations in recent years have altered our conception until now it is generally regarded in the vast majority of instances, as being associated with some organic disease. Many of the cases of hypersecretion occur as the result of gastric or duodenal ulcer, or gastric carcinoma. These may be regarded as the most important intrinsic causes. A considerable group are the result of reflex irritation from disease elsewhere in the abdomen, such as chronic appendicitis, chronic cholecystitis, and gall stones. Other extrinsic causes may produce hypersecretion by reflex stimulation and include organic diseases of the nervous system, particularly tabes dorsalis and to some extent certain functional disorders such as hysteria, psychoneuroses and vagatonia. There are some who claim that in certain instances the hypersecretion must be regarded as a pure neuroses, and this condition may be brought on by mental strain, anxiety, fatigue, overwork, etc. If such cases occur they must be decidedly in the minority and it is far safer to look upon hypersecretion, both the periodic and continuous varieties, as some expression of intrinsic or extrinsic organic disease. The essential feature in the production of hypersecretion is pyloric stenosis. It is therefore an expression of a motor abnormality. Stenosis of the pylorus may be complete or incomplete, due to organic disease or spasmodic, and the extent to which hypersecretion occurs is in direct proportion to the amount of pyloric obstruction that exists. The extrinsic causes of hypersecretion bring about pyloric spasm reflexly; the intrinsic causes act by direct irritation or produce actual anatomical obstruction. It is more than likely that in the many instances both organic obstruction and spasm are associated. The acute exacerbations of the periodic form of continuous hypersecretion are the result of sudden spasmodic closure of the pylorus that is already partially occluded. When the pylorus relaxes the acute manifestations subside. In the chronic continuous form such manifestations do not occur, but the hypersecretion is continued because of the more or less constant motor error that exists.

Periodic Continuous Hypersecretion.—SYMPTOMATOLOGY.—A moderate degree of continuous hypersecretion may go unrecognized until sufficient narrowing of the pylorus induces an acute exacerbation. In these cases a careful history usually elicits symptoms suggestive either of a previous gastric or duodenal ulcer or neoplasm, or the probable existence of chronic disease of the gall-bladder or appendix.

The attack usually begins with a sense of distress, fullness and discomfort in the epigastrium. This soon gives place to pain in the region of the stomach. The pain is often severe and agonizing, and may be more burning or boring in character. The pain is sometimes temporarily relieved by the taking of food or alkalies. The patient shows marked distress, the pulse becomes rapid and weak, and there is marked prostration. Finally vomiting supervenes and relieves the pain. There is a variable period of comfort after the vomiting, associated with various gastric symptoms, until a reaccumulation of the excessive gastric contents occurs, when another paroxysm develops.

The vomitus in this form of periodic hypersecretion is characteristic. The amount of the vomitus is usually very large, amounting to from 100 to 500 c.c. It consists almost entirely of fluid, and the amount of solid material present contrasted to the liquid is small. The proportion of fluid to solid observed by Crohn in his series varied from 4-1 to 10-1. The vomited material contains free HCl and a certain amount of bile is usually mixed with it, giving it a greenish yellow color. The quantity vomited is always noticeably greater than the amount of fluid that has been recently ingested. The vomiting often occurs in gushes.

Rossbach describes a type of period hypersecretion associated with severe migraine. Invariably the sufferers from this form of hypersecretion, complain of severe thirst. The bowels are always constipated, and urine diminished in quantity and concentrated.

Diagnosis.—The diagnosis is made by recognizing the irregularly recurring attacks during which the patient vomits large quantities of acid fluid gastric contents containing little or no solid material. The important feature of the diagnosis is to recognize the underlying cause of the pyloric spasm that is accountable for the acute exacerbations. The possibility of these attacks occurring in tabes dorsalis should not be lost sight of, and this condition should be searched for routinely.

Treatment.—The essential thing in the treatment of the periodic form is to relax the causative pyloric spasm. To this end patients should be put at **rest**. All food and fluid by mouth should be stopped. The stomach should be emptied if possible by **lavage**. **Alkalies** should nearly always be given to neutralize the acidity. Local applications of heat in the form of **hot water bags, electric pads** and **stupes** are of distinct benefit both for relieving the discomfort and for relaxing the pylorospasm. **Atropin** in large doses may also prove beneficial in overcoming the pyloric obstruction. After the acute attack has been controlled, the return to food should be accomplished slowly and with great caution. If the patients are markedly dehydrated, much benefit may result from continuous enteroclysis of salt solution or when acidosis is present, glucose and sodium bicarbonate may be used. The subsequent treatment depends upon the underlying cause of the condition. This in the majority of instances is surgical and each case must be decided on its merits.

Chronic Continuous Hypersecretion.—Symptomatology.—As has been pointed out, hypersecretion is the result of varying degrees of pyloric obstruction, due to some extrinsic or intrinsic cause. As a result, throughout the entire digestive and interdigestive periods, the stomach always contains an excess of secretion, which in a vast majority of instances is hyperacid. The symptoms, therefore, are but a combination of those of hyperchlorhydria associated with varying degrees of pyloric obstruction.

Heart-burn, eructations of acid material and epigastric fullness and pain are characteristic of the condition. The symptoms bear a definite relation to the taking of food, and vary with the quantity and kind of food taken. Sometimes after a meal has been ingested symptoms are in abeyance, and remain so for two or three hours after the meal. It is not uncommon to have the symptoms occur in the early morning. The taking of food or alkalies, or emptying the stomach by lavage, or vomiting, relieves the symptoms. Vomiting is not characteristic of the condition. Constipation is the rule. Remission of the symptoms occur from time to time, and may last several days or a week. As a rule, however, a certain amount of discomfort is experienced at some time during each day. Dietetic errors, over-exertion and fatigue or mental disturbances may bring on acute exacerbations of symptoms.

The condition is recognized by repeated examination of the contents of the fasting stomach. A single examination is not adequate. In this continuous chronic form of hypersecretion, during the fasting period, the stomach contains about 100 c. c. of watery secretion, mixed usually with some food remains and giving a definite reaction to free hydrochloric acid. The best time to carry out these examinations is in the morning, when nothing has been taken into the stomach, not even water, during the night before.

DIAGNOSIS.—This depends upon the examination of the fasting stomach contents. In view of the fact that continuous chronic hypersecretion is secondary to organic disease, the important part of the diagnosis is to find the underlying cause, and in the severe cases it is usually found to be an ulcer, but the other causes such as neoplasm, gall-stones, chronic appendicitis and disease of the central nervous system must not be overlooked.

TREATMENT.—This involves a careful and systematic search for the cause. Until the underlying cause, be it ulcer, appendix or gall-stones, is removed, no hope of a satisfactory cure can be entertained. While the underlying cause is being discovered, or if for any reason radical removal of the cause cannot be undertaken, various measures are often helpful in relieving the symptoms. **Diet** is of importance. When ulcer is suspected some of the well-known diets suitable to ulcer should be employed. In any case it is better to allow but a short time between feedings so that the excess of acid that is present may be neutralized. The suggestions that were made in discussing the diet for hyperchlorhydria are applicable as well to this condition. **Lavage** with either plain water or bicarbonate of soda solution is frequently of great use. In most cases it is sufficient to aspirate the contents of the fasting stomach each morning, but in the more severe cases it is often necessary to empty the stomach several times in 24 hours. So far as drugs are concerned, **alkali combinations** are the most useful, and should be given in sufficient doses to neutralize the acidity.

PROGNOSIS.—In the continuous and periodic forms of hypersecretion, the prognosis depends entirely upon the underlying cause. Until the underlying cause be found and removed, though the treatment may modify somewhat some of the symptoms the condition can never be cured.

Alimentary Hypersecretion.—The alimentary or digestive type of hypersecretion stands out as distinctly separate from those that have just been described. In this type the fasting stomach is empty, but during the period of digestion there is an excessive quantity of gastric juice poured out. It is probable that this form of hypersecretion also is due to some motor disturbance. Some instances have been found with mild degrees of atony and dilatation, but in the majority of instances noteworthy motor defects are absent. Crohn and his associates are inclined to regard alimentary hypersecretion merely as a chemical phenomenon occurring during the ingestion of a meal, and feel that some organic condition, usually an ulcer, is the underlying factor. Many observers, however, are inclined to look upon this type of hypersecretion as a secretory neurosis and not the result of any organic disease. Strauss advanced the belief that in these cases of hypersecretion, hyperacidity existed first, as the result of which starch digestion is interfered with. The undigested starch being retained in the stomach, caused an increased stimulation of the secretory glands, which responded by pouring out an excessive quantity of neutral or alkaline fluid, capable of neutralizing the acid secretion and known as the "thinning fluid." In this way a large amount of secretion occurred at the time of digestion.

SYMPTOMATOLOGY.—Descriptions of the symptomatology of this condition vary considerably. Boas insists that the most important symptom is extreme loss of weight, frequently amounting to from ten to twenty pounds. There is no particular disturbance of the appetite, and there is no evidence of motor insufficiency. A varied array of gastric symptoms occur such as epigastric discomfort, fullness, eructations, sometimes pain and rarely nausea or vomiting. It is found that obstinate constipation is the rule. On the other hand, Lockwood describes cases in which the symptoms resemble those of gastric or duodenal ulcer. The same author has also observed another group of patients whose gastric symptoms are very insignificant and in whom the condition was only discovered accidentally as the result of routine examination of test meals.

In this condition gastric analysis following test meals usually shows a moderate degree of hyperacidity, the total quantity expressed averaging between 100 and 200 c.c. The fluid part of the test meal is four or five times greater than the solid, which tends to settle as a thin lower layer. When motor insufficiency and retention exists, the lower layer of the gastric contents is made up of coarser solid particles and is markedly increased in amount.

DIAGNOSIS.—The diagnosis depends chiefly upon careful examination of the contents of the fasting stomach, and the contents of the stomach after a test breakfast has been administered. The fasting stomach will not show any abnormality in the quantity or quality of its contents, whereas after the test breakfast there will be a definite increase in the amount of secretion, with or without an associated hyperacidity. As has been above noted the gastric contents will have a characteristic appearance.

TREATMENT.—The general condition of the patients must be taken into consideration and every effort should be made to control all emotional and nervous disturbances, and at the same time to have them gain weight. This must be accomplished by **diet**, and by the same means a lessening of the over-abundant gastric secretion may be brought about.

The diet should be an easily digestible mixed one, consisting largely of protein and fat with a minimum of carbohydrate. All irritating highly seasoned food should be avoided, and in some of the severe cases a diet suitable for gastric ulcer may prove of marked value. **Lavage** carried out every day or every second day, with a weak **silver nitrate solution** has been found beneficial. Alkalies, particularly the various forms of **magnesia**, are beneficial not only in lessening the acidity, but also in helping overcome the constipation. **Belladonna** may also be employed. By dietetic and hygenic treatment, the subjective symptoms can largely be eliminated, although it has been pointed out that the actual hypersecretion is rarely removed and may exist for years, even when the patients are entirely well subjectively.

PROGNOSIS.—The prognosis is favorable in most instances. As has been stated, the condition is more likely a secretory neurosis than of organic origin.

CHAPTER XIX

THE EXAMINATION OF THE FECES

By Henry Wald Bettmann, M.D.

Quantity of feces, p. 573; Form and consistency, p. 574; Odor, p. 574; Color, p. 574; Examination of the feces for undigested food-particles, mucus, pus, blood, worms, bacteria, and protozoa, p. 575:—Mucus, p. 575; Pus, p. 576; Blood in feces, p. 576; Worms, bacteria and protozoa, p. 577.

Quantity of the Feces.—A person who is abstaining from all food will nevertheless void a certain amount of feces daily. These feces are composed of the unabsorbed residues of the intestinal excretions. The famous faster, Cetti, during a ten days' fast excreted a daily average of 22 grams of feces containing 3.8 grams of solid residue. The feces were composed of epithelial cells, excretions from the mucous membrane of the intestines and bacteria. When easily digested food is eaten the quantity of the feces increases because of the greater amount of digestive secretions and detritus. When food capable of complete digestion is given, the chemical composition of the feces remains the same whether the food is animal or vegetable. A man fed on a small quantity of perfectly prepared meat will produce the same feces as if fed on perfectly cooked rice. The variation, therefore, in the quantity and quality of the feces in health depends chiefly upon the amount and kind of indigestible residue contained in the food. According to Rubner's table a man fed chiefly on eggs or meat will excrete daily about 64 grams fresh feces; on white bread 109 grams; on a mixed vegetable diet 270 grams; on carrots 1092 grams; and intermediate quantities on various mixtures of animal and vegetable food. Enormous quantities of fecal residue may be retained in the colon in cases of constipation. Schmidt and Strasburger quote Lynch, who records a single evacuation of feces weighing over 20 kilograms (54.20 lbs.). Variations in the quantity of the daily evacuations may have distinct clinical significance. This is especially true if the quantity and composition of the food intake are definitely known, as in the use of the Schmidt test diet. (See below and also Vol. II, pp. 36-38.) When this diet is employed for three days the average evacuation in health is 251 grams of which 54.3 grams are dried residue. In cases of intestinal fermentation the volume rises to 779 grams (129 dried residue); in achylia gastrica with diarrhea, 527 grams (99 dried residue); in obstruction of the bile duct, 944 grams (175.6 dried residue); deficiency of pancreatic juice, 286.8 grams (132 dried residue); severe enteritis, 2780 grams (186 dried residue). In cases of habitual constipation the quantity may be about one-half the normal.

Form and Consistency.—The normal stool is semisolid and sausage-shaped. Soft stools are always pathological. The excessive fluidity is not due to any lack of absorption of water, but to an exudation from the intestinal mucosa. The belief that by drinking much water the consistency of the stools can be changed is erroneous. It is true that feces which remain in the colon too long become dry and hard by the absorption of fluid. The presence of scybalæ '(masses the size of the hazelnut or walnut) indicates that the stool masses have been retained too long in the haustra of the colon. The same is true of the feces resembling sheep dung. Feces of small caliber may be shaped like a lead pencil or a flat band. This does not usually indicate stenosis, but is more often due to functional causes, *i.e.*, spasm of the colon (sigmoid). In many cases in which the diet has been limited for a long time the whole colon may contract to a very small caliber and the feces as a result may be greatly reduced in width. On the other hand, normally formed stools may be passed even in the presence of stenosis of the bowel. The feces may form below the point of stricture and their appearance may give no indication of the presence of an obstruction. The drinking of milk may cause the stools to have a peculiarly resistant or putty-like consistency which may lead to impaction.

Odor.—The odor of the feces may reveal important clinical facts. Sour-smelling stools almost always indicate the occurence of an abnormal fermentation of carbohydrates within the intestine. In these cases the stools are apt to be light in color and frothy, and form bubbles on standing. A strong putrefactive odor is evidence of decomposition by bacteria of excess proteid material; these stools are apt to be dark in color. In Asiatic cholera the stools may be odorless, owing to the very rapid passage of the fluid through the intestine. Smithies calls attention to the peculiar odor of the stools in certain protozoan infections of the intestinal tract. He describes it as a peculiarly pungent, penetrating, musty, sometimes definitely ammoniacal smell which may be often putrid and disgustingly nauseating. Quincke and Roos have compared the odor of the stools in entamebic dysentery to glue. After gastric or duodenal hemorrhage the tarry stools which follow usually have a disgustingly foul odor.

Color.—The color of the feces is subject to many variations. In health the coloring matter of the bile undergoes a gradual reduction in the lower ileum and colon and becomes hydrobilirubin which gives to the feces its normal brown appearance. If a small mass of feces is well mixed with a concentrated solution of bichlorid of mercury and allowed to stand for twenty-four hours, the color changes to a bright red (Schmidt Sublimate Test. See Vol. II, p. 40). When bile is excluded from the intestine (obstructive jaundice), no change of color occurs. When the stool turns green (biliverdin) with the sublimate test, it indicates a too rapid passage of the feces through the intestine or an interference with the natural reducing process. The acholic stool owes its gray or white appearance (clay-color) also to an abnormal admixture of fat. Light-colored stools in which excess of fat is a factor occur also in pancreatic disorders, in sprue, tuberculosis of the intestine and disease of the mesenteric glands. It is possible that under certain conditions the formation and flow of bile may be temporarily suspended leading to deficient staining of certain portions of the feces; and in other cases the bile pigment may be changed into the colorless leukourobilin instead of the normal hydrobilirubin.

Diet and drugs have a great effect on the color of the stools. On a milk diet the stools become very light yellow. The feces of meat eaters are darker than those of vegetarians, owing to hematin. The color is affected by green vegetables, blackberries, cocoa, and red wines. Various drugs have a striking influence. Iron and manganese give a dark greenish black color; calomel a green hue; bismuth an almost black tone. Santonin, rhubarb and senna color the stools yellow or yellowish brown in varying degrees. Catechu may give a reddish tinge which may look like blood. Blood itself, if from the colon, is red in appearance. If it comes from the small intestine, duodenum or stomach, it is usually black or tarry when voided. This dependable rule has two exceptions. Blood from the small intestine may be so rapidly voided and in such large quantity that it retains its red appearance. Blood originating in the colon may be retained long enough to appear dark when passed.

Examination of the Feces for Undigested Food-particles, Mucus, Pus, Blood, Worms, Bacteria, and Protozoa.—The feces of normal persons on a substantial mixed diet always contain food remnants. The bulk of the feces depend almost entirely upon the amount of indigestible material which is consumed. Most of the coarse vegetables have little food value compared to their bulk; their usefulness is chiefly to supply "roughage" to incite intestinal peristalsis. In every normal stool, therefore, on a mixed diet we find many vegetable fibers, imperfectly broken down cellulose structures containing starch granules (free undigested starch granules are not found in health), fruit pits and skins, fatty acids and soaps, a few muscle fibers, and sparse elastic or connective tissue fibers.

MUCUS.—Mucus must always be considered pathological but its clinical significance varies greatly under different conditions. Just as in coryza, a mucous discharge from the nose or throat may indicate simply a passing irritation, so the transient appearance of mucus in the stool may also be a sign of a merely temporary irritation, and mucus may appear after a strong physic or exposure to cold, and may be present in cases of simple diarrhea. Mucus evident to the naked eye almost always originates in the colon. Small flocculi of mucus in fluid stools may be suspected as coming from the small intestine when they are seen under the microscope to contain granules of bilirubin and nuclei or small-cell remnants. As Schmidt has pointed out, the amount of mucus need not bear any direct relation to the degree of irritation or inflammation. In attacks of mucous colitis, huge masses may be discharged without any inflammatory changes to speak of; in membranous colitis inflammation may be scanty or absent. The degree of inflammation may usually be gauged by the number of leukocytes adherent to the mucus; as a rule, the greater the number of leukocytes, the greater the inflammation. In intense inflammation, erythrocytes may occur with the leukocytes and this fact is often obvious to the naked eye (bloody tinge). Epithelial cells clinging to the mucus are almost invariably of the columnar type. They are often present in fairly large numbers but their clinical significance is slight.

PUS.—Small round cells (polymorphonuclear leukocytes and pus cells) do not occur in normal stools. Their presence always indicates inflammation. Pus, recognizable as such by the naked eye almost always originates in the lower portions of the colon or rectum, but it may indicate the rupture of an abscess into the bowel. Pus may occur in dysentery, cancer of the bowel, syphilitic or benign ulceration, and rarely in the acute exacerbations of proctitis and chronic colitis.

BLOOD IN THE FECES.—The commonest source of blood in the feces is the rectum. Hemorrhoids and fissures are the most frequent causes. Cancer is next in frequency; polyps are more rare. In cases of fissure and hemorrhoids the blood is apt to occur in streaks adherent to the feces. In severely bleeding piles, however, the evacuation of feces is often followed by a free discharge of blood which may stain the toilet basin bright red. The bleeding may be so slight that it escapes the naked eye, but red cells can be recognized under the microscope. In cases of polyposis the stools are slimy and the blood is intimately mixed with them. In cancer, the blood is usually accompanied by mucus and the frequent evacuation of blood and mucus without feces should arouse suspicion of malignancy, although chronic ulceration may produce the same result. In dysentery, the blood may be so intimately mixed with the fluid stool that it gives it the so-called meat-juice appearance.

When hemorrhages occur in small quantity and high in the digestive tract, the blood will escape observation with the eye and the microscope, and can be detected only by chemical tests.

The chief value of the chemical test for so-called occult blood is in the diagnosis of ulcer or cancer of the digestive tube. When cancer of the stomach or intestine begins to bleed, occult blood can be detected constantly in the feces. In cases of ulcer, bleeding is apt to be intermittent or absent and the negative test is of relatively little value. After a known hemorrhage, however, the progress towards recovery can be estimated by the gradual cessation of bleeding. A positive reaction is reliable only if all sources of hemorrhage such as the nasal mucous membrane, the gums, the rectum and the anus can be absolutely excluded and if all food containing blood (meats, fish, soups) has been omitted from the diet for two, or better three, days before the feces are examined. The stool should be as fresh as possible. The best clinical test is that with benzidin after the manner of Schlesinger and Holst. A small portion of feces is rubbed up with distilled water in a test tube and brought to the boiling point to destroy ferments. In a second test tube a concentrated solution of benzidin in glacial acetic acid is prepared. To 2 c.c. (32.4 minims) of this solution is added 3 c.c. (48.6 minims) of 3 per cent. hydrogen peroxid. This solution should remain colorless. To it are added a few drops of the feces mixture. If even minute traces of blood are present, a green or blue green ring is formed. Other tests are also reliable and especially to be recommended are Schumm's aloin test. Benzidin tablets and benzidin paper are also on the market for rapid testing by the practitioner without a laboratory.

WORMS, BACTERIA AND PROTOZOA.—For the study of the feces for worms and their eggs the reader is referred to the appropriate sections of this work. The study of the stools for amebæ, the flagellates, the bacilli of typhoid fever and dysentery has been most exhaustively described and discussed in Volume IV, Chapters xxxi to xxxix. All the pancreatic function tests have been thoroughly described in Volume II, Chapter xiii, and the reader is referred to these sections.

CHAPTER XX

ACUTE, CHRONIC AND ULCERATIVE ENTEROCOLITIS

BY B. B. VINCENT LYON, A.B., M.D.

and

HENRY J. BARTLE, B.S., M.D.

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Foreword.—Before outlining the various inflammatory conditions of the acute, chronic and ulcerative states of the large and small bowel, we desire to present for consideration certain underlying fundamental principles of intestinal function which, if understood, will greatly facilitate your conception of bowel dysfunction; for, after all, the various symptoms that result from bowel disease are largely those of mechanical, biochemical and bacterial defects.

We must first conceive of the bowel, large and small combined, as a transit tube through which are propelled food, liquids and gasses, changed or unchanged from the stomach to and through the anus. The propulsion is effected by action of the bowel musculature, the contraction of which produces various effects on the size and shape and position of a segment of gut, such as narrowing of the lumen, shortening of the segment and imparting a screw-like motion.[1] And because of these motions occurring in several segments simultaneously the relative positions of the various loops of small bowels are constantly being shifted. This muscular activity, under normal conditions, takes place in a regularly recurring rhythm. As shown by Alvarez [2, 3] it begins in the stomach fundus and spreads over the stomach to the pyloric ring. Beyond the pylorus it begins again and extends down along the small bowel. It becomes more intense at certain points (Nodes of Keith)[4] where it is augmented and given fresh impetus and finally spreads down over the large gut. In the stomach there may be several small contractions occurring in rapid sequence, as visualized by the fluoroscope, but as the lower small bowel is reached the contractions become less numerous but more powerful until the colon takes up the wave. The farther down the colon we go the less frequent are the waves, but they take on increasing intensity so that the cecum and ascending colon, while always undergoing some motion, may only pass their fecal content onward every thirty minutes or even less frequently. A massed action has been referred to in the transverse colon in which the whole content is suddenly shifted by a powerful contraction every six hours or so. Finally the pelvic colon (iliac portion of the descending and the sigmoid) may go into very active peristalsis only once every twelve or forty-eight hours and expel its contents through the rectum and anus. We style this process defecation. You will note that we say through the rectum, for, contrary to the usually accepted belief, the rectum is not a reservoir but only a vestibule and should normally be empty; the fecal mass being physiologically restrained from its entry into the rectum by O'Byrne's sphincter, constituting the rectosigmoid apparatus.

All this motion of contraction or dilation, shortening or lengthening, torsion and vermiform is under the control of two systems of nerves, parts of the vegetative nervous system. First, we have the sympathetics, which are easily remembered as "the brakes" of the gastrointestinal tube, for their function is largely one of inhibition. The second are the parasympathetics (vagus and pelvic) which convey the motor and secretomotor impulses to the muscle fibers and mucosa through their two plexuses, Auerbach and Meissner, situated respectively in the muscular wall and submucosa throughout the canal. These two nervous systems are in turn controlled by plexuses situated along the anterior surface of the vertebral column from the skull to the coccyx and are supplied by the spinal and cranial nerves. The bowel tube is lined by a secreting and absorbing layer of epithelium and is covered over the outside with a fine serous membrane (visceral peritoneum) and finally, and not least important, through its mesentery it receives its arterial, venous, lymph and nerve supply. Therefore, we have a rather complicated flexible tube with which to deal and many things may go wrong with it, but we must keep in mind the fact that sensory nerves are not distributed to this viscus except at the anus and possibly in the mesentery and that *pain* may, as a result, be only a *late* symptom of disease; whereas *errors of motility* are likely to be *early* symptoms. The presence of solids, liquids or gas within the tube is enough to stimulate peristalsis, for it is an inherent function of organs constructed of unstriped muscle (and striped in the case of the heart) to empty themselves after once being distended. So the bowel contracts down on its contents and naturally pushes, squeezes or cork-screws it onward. But errors in motility occur [5] and overstimulation at one of Keith's nodes may bring about a tonic contraction of the bowel at this point, preventing the onward passage of the bowel contents, being brought down from above, with a consequent distention of the bowel wall. This stretches the plexus of Auerbach and pulls the bowel mesentery from its normal position and thus pain is reflexly produced in the abdominal wall (perhaps rigidity too) whence it is referred by the spinal cord.[6] Or, if disease extend through the muscular coat and irritate the parietal peritoneum, since this is supplied with sensory nerves, pain results. Thus you will see that errors of motility give most of the subjective symptoms, such as pain, fullness, pressure distress, borborygmus, diarrhea, constipation, nausea, and vomiting. And, too, these same defects in motility may give some of the objective findings, atony, bloating, spasticity, hypo- and hypermotility.[7] Then the *errors of secretion* have to be remembered and here we have two factors to consider. First, due to improper innervation of the glandulature, we may have hypo- and hypersecretion. And second, due to disease of the lining of the mucous membrane and, depending upon the extent of this process, involving the deeper layers of the bowel, we may have an outpouring of mucus from simple irritation, or, in atrophy, a paucity of enteric juices or mucus. Where superficial or deep necrosis has resulted, pus and blood in varying amounts and exfoliation of necrotic structures will occur.

From a *biochemical standpoint* we must take into consideration the nature of the juices elaborated by the gastric and enteric glandulature as well as the auxiliary digestive glands (salivary, liver and pancreas). This secretory function may be impaired permanently or suppressed temporarily or it may be transiently or constantly overactive. Here again, nervous impulses may be responsible for these variations and this nervous dysfunction in turn may be the result of improperly acting or diseased endocrines (pituitary, pineal, thyroid, parathyroid, adrenal, ovary, testicle, pancreas, spleen). Or the digestive organs themselves may be the seat of actual pathological change, thus altering their power of or type of secretion.

Again the question of the *bacterial flora of the gut* must be considered. We know that as we descend the small bowel the number of bacteria, some native and useful and some alien and harmful, increases until the terminal ileum and cecum are reached. At these points the growth of flora is most prolific and as we descend the large gut the number is again lessened until the rectosigmoid region is reached where the bacterial flora becomes less intense. Not only the number of bacteria but the types present must be noted: saprophytic or parasitic, aërobic or anaërobic, Gram positive or negative staining, cocci, streptococci, staphylococci, bacilli or streptobacilli, putrefactive and fermentative types. Therefore in studying a patient it is necessary to have some conception of normal function and excreta.

To check up on motility we have two methods at our disposal. The first, which is the easiest, consists in giving the patient 10 grains (0.65 gram) of carmin in capsules and timing its appearance and disappearance at the anal outlet and observing the manner in which it is mixed with the stools, *i.e.*, whether intimately or if it just colors portions of several stools. The color normally should appear within twenty-four and disappear in about forty-eight hours from the time of taking, and should color partially or completely one to three stools. The type of chart which the authors give their patients and the directions for taking the carmin are given on pages 481 and 482.

The second method for determining motility is by the roentgen rays and from the time of drinking the barium, the normals usually accepted are as follows: Stomach begins to empty at once and should be empty within four to six hours; cecum is reached in from two to three hours, and all barium should have passed through the ileocecal valve in from five to ten hours; hepatic flexure should be reached in six hours and cecum and ascending colon should be empty in from eighteen to twenty hours (Manges), or from four to thirty-six hours (Brown and Sampson)[8]; splenic flexure reached in from six to ten hours and transverse colon empty in from twenty to twenty-four hours; sigmoid reached within twenty to twenty-four hours and complete discharge should have occurred in from twenty-four to forty-eight hours (Manges).

The *normal* appearance of the stool should be a formed smooth cylinder, four to eight inches long, and in cross section the size of our quarter or half dollar, of a decided brown color (depending largely upon the diet, pale in vegetarians except after spinach, very light where much milk is drunk, and dark after ingestion of meats, etc.), neutral in reaction, odor neither acid, sour, pungent nor putrid, it should usually sink in water, and should show grossly no mucus or undigested food particles. Microscopically, we see some connective and elastic tissue, often meat fibers, but usually devoid of striations and nuclei, no free fat or fatty acid crystals, but we usually see soaps and at times soap crystals. There is usually vegetable residue, but no free starch cells; crystals of ammonium magnesium phosphate or triple phosphate, calcium phosphate and calcium oxalate may be seen. No mucus or red blood-cells but a few leukocytes and desquamated epithelial cells are frequently seen.[9]

ACUTE ENTEROCOLITIS

Synonyms.—The synonyms of acute enterocolitis fall into the following group: Simple acute catarrhal enteritis, enterocolitis, or colitis;

OFFICES OF
B. B. VINCENT LYON, M.D.; HENRY J. BARTLE, M.D.,
PHILADELPHIA.

· INSTRUCTIONS FOR PATIENT UNDERGOING A DIAGNOSTIC STUDY.

In a diagnostic study of your case, such as we are undertaking, certain things have to be done by us to find the cause of your illness. To do these things well, and to obtain the correct results, we have trained ourselves and our assistants in various methods of investigation which we have found give the least discomfort and inconvenience to the patient without lessening the value of the procedures from a diagnostic standpoint. However, much depends on the willingness and faithfulness with which you carry out the instructions, as to whether success or failure comes of our efforts. Therefore, please bear in mind that the following instructions are given for a definite purpose, and are actually what we require you strictly to do. Furthermore, do not try to memorize them, but consult them at 6 P. M. of each day while you are under study, to learn what preparation you are to make for the following visit.

1. You will be given a jar (which you will label with your name and date), a basin and two wooden blades. In the jar you will bring on...............morning the last stool passed by you before coming to the office. It is well to collect the stool the day before coming, and if you should have a subsequent one before starting out for this office, discard the old one, wash out the jar thoroughly, and place the latest one therein. Pass the stool directly into the basin, and with the aid of the two wooden blades place all of the stool within the jar, carefully avoiding smearing the outside of the jar, and adjust cap down snugly. Keep the jar in the coolest place you can find. Urine or menstrual discharge should not be allowed to mix with the feces, as they will cause errors in the examination. If you are given a special diet by us to follow before bringing the stool, please give this your most careful consideration, and do not eat anything that is not printed on the diet slip.

2. In one of two bottles you will bring a specimen of urine collected two hours after your heaviest meal of the day. Keep it cool. Just before collecting this sample, the genitals are to be washed with soap and water as thoroughly as possible, so as to prevent anything except urine entering the bottle. The following morning,..............., at 7 A. M., please pass your urine, cleansing the parts again, and bottle a portion of it and label it "A. M." and bring both these bottles with you. Write your name on each bottle. Drink a glass of water at 7 A. M. and again at 8 A. M., but do not pass urine again until you get to the office at 8.45. You may eat your regular breakfast this day.

3. At 9 P. M. on.........................., and again on ....evening, you will please eat one meat sandwich, with twenty raisins or currants, and drink a glass of water or hot milk and water mixed and sweetened. Eat or drink nothing after this either during the night or early morning until we give it to you in this office at 8.30 A. M. Do not brush your teeth, and do not swallow saliva that may form in your mouth or mucus that may be coughed up or brought down at the back of the mouth. This is very important.

4. The two red capsules in the envelope contain a dye which is intended to color one of your meals as it passes through you, so that we may be able to ascertain how long any one meal stays in your digestive tract. Swallow these two capsules with the *next meal after* a bowel movement on.................... Every bowel movement after taking them is to be watched by you and recorded on the inside of this sheet in its proper column until no more red is to be seen in the stool. Have the stool passed into a pan or chamber, and examine it in a good light, and then finally pour water into the vessel to observe whether the stool floats or sinks, being sure that it is not stuck to the bottom.

The order of these examinations will be:

Chart Given Authors' Patients and Directions for Taking Carmin.

NAME, DATE, HOUR, A. M., P. M.

THIS REFERS TO TIME OF TAKING CAPSULES

Description Bowel Movements. DATE and HOUR A. M. or P. M.	1st	2nd	3rd	4th	5th
COLOR—red, brown, red and brown, yellow-brown, etc.					
FORM—sausage, small, balls, long string, pencil, flattened ribbon, mushy, liquid.					
AMOUNT—roughly in teacupfuls, tablespoonfuls, etc.					
MUCUS—Jelly-like masses, skin-like strings, or sticky and glistening.					
UNDIGESTED FOOD— Hulls of corn, beans, peas, seeds or fruit skins.					
ODOR—Sour, putrid, pungent, very offensive.					
FLOATS OR SINKS— Completely cover with water; shake free of sticking to bottom.					

SUMMARY, (Do not write on this line) Appearance, Hours. Disappearance, Hours. Laxative?

REVERSE SIDE OF CHART FOR CARMIN TAKING

acute duodenitis, jejunitis, ileitis, ileocolitis; acute diarrhea; acute intestinal catarrh; acute intestinal indigestion; acute enteric intoxication; cholera infantum; dysentery; and cholera morbus.

Definition.—Acute enterocolitis is a circumscribed or diffuse, acute inflammatory process, involving the small, small and large, or large gut alone.

Etiology.—Certain *predisposing causes* manifest themselves in children in such form as scurvy, rickets, malnutrition, syphilis, tuberculosis, and dentition. These may be important factors in the production of recurrent or even primary attacks of enteritis.[10] Seasonal variations, such as extreme heat or sudden drops in temperature, and filthy surroundings, as well as burns, nervousness, and visceral ptosis are contributing factors. Here, too, must be mentioned achylia gastrica in which there is no free hydrochloric acid being elaborated by the stomach mucosa. Because of this the proteids are not converted into peptones, but are thrown out directly into the duodenum and if the pancreatic juice is deficient, they may act as irritants. *Primary causes* are: Cholera, dysentery (amebic and bacillary), typhoid fever (*q. v.*); bacterial invasion by streptococci and staphylococci pyogenes, and bacillus pyogenes, bacillus coli, bacillus pyocyaneus, bacillus Shiga, bacillus Flexner, bacillus aërogenes capsulatus (Welchii) and bacillus Morgani,[11] as well as certain amebæ; toxins resulting from bacterial growth in infected milk, which, even though the milk be sterilized before use, may remain; leukomains resulting from the fermentation and decomposition of food; ptomains in tainted meats, fish and shell-fish; idiosyncrasy to certain foods such as the crustaceans, berries, oysters, etc., larvæ of flies; in babies, improper modification of milk, excessive and too frequent feedings; large amount of ice-cold drinks, ice cream, fruit ices; excessive amounts of indigestible foods with large cellulose residue; large amounts of nuts, candies, spoiled filling of cream puffs, etc.; foreign bodies; and medicinal irritants, as mercury, arsenic, drastic purgatives, and corrosive poison. Acute enterocolitis may also occur secondary to other bodily infections such as influenza, pneumonia, scarlatina, tonsillitis, measles; and to constitutional disorders such as Bright's disease, uremia, diabetes, heart and lung diseases producing passive congestion, anemia, cirrhosis of the liver and malignant disease causing portal congestion.

Symptomatology.—MILD CATARRHAL INFLAMMATION.—The symptoms of acute enterocolitis are dependent upon two things: First, the type of infection or irritation; and second, the portion of the bowel involved and the degree of involvement. The mildest type of enteritis is presented in a low-grade duodenitis or the extension of a gastritis. In this type we usually have a history of overeating or overindulgence in drinks, usually of ice drinks, of having eaten an unusual type of food or a combination of many different foods, of excessive indulgence in sweets or highly seasoned articles of diet, or perhaps of bolting the meals while under mental stress. The individual complains of an epigastric sense of discomfort, fullness, weight, or pressure, perhaps some burning and later nausea and mild fever. Should the nausea lead to vomiting within one or two hours after the meal, complete relief may be obtained with care in the diet or abstinence from food for one or two days and there may be no further symptoms. If vomiting does not occur and the irritating substance is allowed to enter the small bowel in large amounts, then a greater involvement of the small bowel results. In this form there will be fullness and distention of the abdomen, borborygmus, and perhaps the passage of foul flatus with periumbilical tenderness or a moderate temperature and diffuse abdominal colicky pains may be the only signs of an enteritis. A mild diarrhea may supervene within twelve or twenty-four hours independently of, or coincident with the self-administration of a laxative. A slight degree of jaundice may often be observed in a subicteroid hue of the sclera or of the mucous membrane covering the palate. The jaundice is due to a partial blocking of the common duct at its entrance into the duodenum and becomes apparent in from twelve to twenty-four hours. The papilla of Vater is swollen and edematous, thereby obstructing the outflow of bile from the common duct. A jaundice developing later than one or two days after the onset of an attack (acute catarrhal jaundice) is due to extension of the inflammatory process to within the common duct. The tongue may be coated, and usually there is a fairly well-marked anorexia. Here, too, with a complete evacuation of the bowel and care in diet for a few days no further symptoms may develop.

If the ingested material is more irritating and is not removed from the bowel in a short enough time, or if the patient's resistance be lessened to a greater degree than in the two former instances, an ileocolitis may be set up. If the inflammatory process extend to the transverse and descending colon, the sigmoid and the rectum, then to the symptoms above described we will have added a marked diarrhea and increased thirst. This, too, is accompanied by colicky pain preceding an evacuation. The range of temperature is likely to be higher, even as high as 104° F. (40° C.); prostration is greater; and malaise, headache, complete anorexia, nausea and vomiting may be present. This is likely to run a more protracted course than a simple gastro-enteritis. The abdomen is apt to be retracted rather than distended (which we may see where the small bowel alone is involved); hyperperistalsis may be visible and distinctly audible. Tenderness is not localized to the periumbilical region as it may be in simple enteritis, but is usually well marked out into the flanks and along the course of the great bowel.

The degree of diarrhea depends upon the location and amount of involvement of the colon. Involvement alone of the head of the colon or cecum and ascending colon may give very little diarrhea, perhaps some mushy stools, but when the splenic flexure, descending colon, sigmoid, and rectum become involved, the farther down the involvement and the more intense it is, the more the diarrhea. In this simple catarrhal type the first few movements may be only fecal matter, thinner than usual and preceded by griping pain. Later on the feces, if formed at all, will not only contain intimately mixed mucus, but will be coated over with a layer of thin mucus, and after the canal has been emptied of fecal matter the movement may consist of a watery, colorless and odorless discharge, or it may become gray, green or yellow. In children, it may result in the rice-water type of stool. Or thin to semi-jelly like masses of brownish or greenish stained foul-smelling mucus may be the only thing passed. The foulest stools are those resulting from dietetic disorders. If the rectum is involved right down to the internal sphincter, to the diarrhea may be added tenesmus, shown by immediate desire to defecate as soon as the patient has been to stool. The result may be that a small insignificant amount of mucus may be discharged with little or no relief.

In the simplest form of duodenitis or high enteritis the examination of the stool described above may disclose nothing chemically abnormal. If the bowel be involved pretty well down to the caput coli, a smear of the stool on a slide is likely to show small masses of incorporated mucus. It has been claimed that these sago-like bodies are frequently remains of vegetables, but the authors believe that they are actually masses of mucus. The stool itself may have a mirror-like surface due to the intimate mixture of mucus, and is usually sticky and adheres to the vessel. It may be soft and foul-smelling. Rarely do we find occult blood. Food particles may be observed grossly and microscopically. The farther down the involvement occurs the more hurried is the motility. When there is an actual diarrhea in process we may see gross particles of food (lienteric diarrhea), and, if the stool be formed, it will not only have the mucus thoroughly incorporated as in the higher type of catarrh, but it will be covered over with the slimy mucoid material described above, derived from the mucosa of the lower bowel.

With involvement of the larger bowel there is a greater possibility of bleeding. In this instance the farther down the bowel the bleeding occurs, the less likely we are to find the blood within the bowel movement itself. The mucous coating is likely to be blood-stained, or if scrapings are made from the outside of the formed masses (when they exist) and the *test for occult blood* is applied, we are likely to get positive results. This test is best applied by mixing a pea-sized piece of feces with glacial acetic acid to form a thin gruel-like mixture. This may or may not be boiled, and with this there is thoroughly mixed an equal quantity of hydrogen peroxid. Then two drops of a freshly prepared saturated solution of benzidin in glacial acetic acid are added. A deep blue-green color resulting at once denotes blood present in the form of hematin or hematoidin. Instead of adding the benzidin solution, an alcoholic saturated solution of guaiac may be dropped in. This test is much less delicate and is positive if a robin-egg blue color results within five minutes. When positive it definitely indicates the presence of a pathological amount of blood. Tests for unchanged bile as applied by the Gmelin test will usually be positive where the small bowel is involved with a resultant hurried motility through this portion of the gut. On mixing equal quantities of fecal matter and a 25 per cent. solution of bichlorid of mercury (Schmidt test) in a porcelain dish, unchanged bile (or bilirubin) will be demonstrated by the presence of a green color, whereas ordinarily in normal movements the end color reaction is red (hydrobilirubin or urobilin).

Microscopically the stool shows, besides free mucus and incorporated mucus, fatty degenerated epithelium, crystals of triple phosphates and calcium oxalate, food débris, and many bacteria; red blood-cells may be present and, if the process has gone on to ulceration, white blood-cells in enormous numbers constituting pus will be found.

The urine is frequently of high specific gravity when the diarrhea is marked and, with extensive involvement of the smaller bowel, indican is frequently found to be much increased. Rosenbach's reaction for bile may be positive and acetone may be present. Microscopically we may see hyaline casts and a very moderate amount of albumin.

In children the mild catarrhal inflammatory lesions of the small bowel include the acute enteric intoxications and dyspeptic diarrheas. These are frequently described under various distinct headings, but clinically they are mild or intense stages of one and the same condition. An unusual article of food or some other cause, as cited in the foregoing etiology, may produce an illness of sudden onset, in which the child is completely prostrated or restless, with slight fever. There may be nausea and colicky pains, with symptoms of meningeal irritation occasionally.

It is sometimes difficult to make a diagnosis in the child without the history of vomiting or diarrhea. Convulsions may usher in an attack, or the child may be somnolent. Associated with this may be complete anuria extending over a period of twelve to twenty-four or more hours and a condition simulating the so-called uremia of the adult may be noted. History is perhaps the most important means of arriving at a diagnosis. Evacuation of the stomach and bowel may bring about a complete change within a few hours in the child's general condition. Examination of the abdomen may show nothing except some distention. Lumbar puncture gives certain information but in a negative way, as the cell count is not increased and bacteria are not found. As the bowel movements begin to appear, the diagnosis is made evident by the finding of mucus and putrid or fatty acid materials and undigested food in the stools. In these intoxications a paresis of the bowels may be difficult to overcome, so that the question of intussusception or ileus may be difficult to divorce from our diagnosis.

MORE INTENSE TYPES OF ACUTE ENTEROCOLITIS.—Into this classification fall the more virulent infections and ptomain poisonings in adults and the follicular types chiefly in children.

The symptoms in general are about the same as those described in the milder form except for the increased severity and rapidity of their onset. Within four to six hours after the ingestion of the irritant, vomiting and prostration, pain and perhaps tenderness, rapid and high elevation of temperature and diarrhea will frequently follow one another in quick succession. These individuals are distinctly sick. Pain may be so intense as to actually cause a state of shock. In these cases one thinks of acute perforations and the colic of cholelithiasis and nephrolithiasis. The vomiting may be extreme, so that nothing is retained by the stomach. The purging may be so profuse as to cause a marked extraction of fluids from the tissues of the body. Thirst becomes extreme and is a difficult thing to deal with. As soon as anything to drink is given, it is promptly vomited.

The constitutional symptoms, as represented by high fever, prostration, and general bodily discomfort and headache, are marked. The tongue is furred and the pulse more rapid and weak from loss of blood volume. The stools at first are foul and copious, and later watery and frequently of large volume until depletion is well advanced, when they become bloody and mucoid. This represents the picture we see in cholera morbus or ptomain poisoning and its counterpart cholera infantum in children. This is a rapidly developing and intense inflammatory state, usually of the whole gastro-intestinal tract. With proper and early treatment and care of the diet, recovery usually takes place within from one to three or four weeks, with a gradual lessening of abdominal distress and a disappearance of the mucus in the stools.

In the follicular enteritis, seen more frequently in children, the process is likely to be more protracted, running well over four or five weeks and into the chronic types of enterocolitis. The stools do not tend to clear up, the diarrhea does not tend to subside, and all types of nourishment seem to produce irritation and maintain the condition in an active state. Vomiting here is less common than in the more intense inflammation of cholera infantum. There is not the extreme amount of serous diarrhea. The stools contain more mucus and they may run up to twenty or thirty a day and are usually small and frequently bloody and sometimes odorless.

This is a slow process and the child loses weight constantly. The symptoms are those of an involvement of the ileum and colon, rather than of the upper portion of the small bowel. This is frequently designated marasmus.

Diagnosis.—In the *mild* inflammations the diagnosis is usually easy except *in children.* Here, as pointed out above, the history is perhaps the most important aid. We have to differentiate between acute enterocolitis and a beginning pneumonia accompanied with intestinal paresis, where the diarrhea has not already begun. A careful physical examination of the lungs may reveal the signs of pneumonic involvement, but since the early signs of pneumonia in a child are frequently not those of consolidation, giving the usual impairment of resonance and intensification of breath sounds as in the adult, and since there may be only hyperresonance over the lung which is beginning to be involved, with no râles, a physical examination of the chest in a child frequently may not be conclusive. For this reason it is difficult to differentiate between a beginning pneumonia and acute intestinal intoxication. The prostration may be about the same in the two conditions. Usually in pneumonia you have an early temperature of over 103° F. (39.44° C.) whereas in acute intestinal intoxication this may be slower in reaching the high figure, although it is not impossible for it to be up around 104° F. (40° C.) or more within several hours.

Appendicitis in its early stage may be confused with acute enteritis, but constipation is marked in contrast to diarrhea and within a short time the pain begins to localize in the right lower quadrant and the right rectus becomes rigid. Intussusception and ileus must be ruled out.

Typhoid fever is not a frequent disease in children. Its onset is somewhat protracted (but not to the same extent as in the adult), whereas acute intestinal intoxication or inflammation in a child is usually a very rapid process in comparison.

If vomiting be a marked sign and accompanied by fever, one must think of scarlatina and other exanthems, but the examination of the tongue, throat, and buccal mucous membrane, as well as the skin of the body, will clear up the diagnosis. The circumoral pallor of scarlatina is frequently seen in acute enteritis and enterocolitis, especially if diarrhea has started and has become somewhat intense, or at least enough to deplete the patient. In children, when the *more severe form* runs a more protracted course, typhoid of course will perhaps be most logically thought of, but in these cases the temperature is never a typical typhoid temperature, never reaching the height or tracing the course of that seen in typhoid. Rather is it more like the hectic temperature of tuberculosis, with its late afternoon and evening rise and morning low point, or the reverse of this can be seen—morning rise, evening fall.

In the adult the *mild* types of enterocolitis are easily diagnosed, the main point in a diagnosis being as a rule to determine the portion of the gastro-intestinal tube involved. Here again, in cases which run for more than a week, typhoid is to be considered. The temperature in typhoid in contrast to the typhoid temperature of children, is more likely to be typical, with its slow, steady rise, evening after evening, higher and higher for a week, with morning lower than the evening, but higher than the morning before; and then the maintained temperature of from one to three weeks, with its lysis; it is never seen in enterocolitis. While these cases may run along for from one to four weeks, it is rare for them to do so. It is usually an evanescent disease in the adult, rarely lasting more than one week. Of course the Widal test of the blood should be made, the spleen palpated for increase of size, and the skin inspected for the typical rose spots.

There is practically no acute abdominal disease that simulates the milder forms of this catarrh. However, when the *more intense type* of inflammation presents itself there are several conditions which have to be considered and ruled out before an actual diagnosis can be made.

The first as pointed out in the symptomatology, perhaps, is gastric or intestinal perforation. Almost the same shock and collapse may be seen in these two conditions, the moist skin, the violent pain, and vomiting, before the diarrhea has begun. However, careful palpation of the abdomen is the best guide to a diagnosis. There is no rigidity of the abdomen of any marked degree in acute enterocolitis. Between pains the muscles are relaxed. This we never see in perforation. A septic type of diaphragmatic pleurisy may give pain somewhat suggesting acute enterocolitis, but this pain is more likely to be steady, intensified by breathing or coughing and will frequently show the same spasticity of the abdominal muscles as an acute peritonitis from perforation.

Cholelithiasis is sometimes difficult of differential diagnosis. It may or may not have the same late jaundice as enterocolitis, has the same paroxysmal pain but it is usually more severe; the patient does not develop his diarrhea, as usually occurs early in an intense inflammatory condition of the bowel, but may give the same history of heavy eating three or four hours before the onset of the attack.

Nephrolithiasis usually will show bloody urine, but this diagnostic point must not be relied upon exclusively, as the authors have seen cases of nephrolithiasis in which urinary blood could not be found in the first twenty-four hours. The pain here is most often located in one or the other renal regions with its usual characteristic radiation into the pelvis of the female and testicles of the male. Again diarrhea in this condition is rare, as constipation is the more usual accompaniment in kidney stone.

Acute hemorrhagic pancreatitis is to be thought of, but in this condition the prostration is usually more marked and tenderness is likely to be limited more to the mid epigastrium. The diarrhea may be the same in each, but is usually not so severe and may be absent in pancreatitis and as a rule we have a long history of digestive disturbances extending back for several years.

Acute poisoning by antimony, arsenic, colchicum, and fungi may closely simulate the rapid fulminating type of gastro-enteritis and should always be considered where one is called upon to see a severe case of what is ordinarily called cholera morbus.

Thrombosis and embolism of the inferior and superior mesenteric arteries and veins and of the portal vein must be ruled out, as immediate operation may have to be performed to save the patient's life. (Refer to section on Embolism and Thrombosis.)

Asiatic cholera and likewise uremia must be thought of.

Nervous diarrhea must not be confused with acute enteritis. Here we have a history of a nervous shock and the symptoms are the result of an acute vagotonic stimulation of peristalsis, subsiding promptly after the thorough emptying of the bowels.

Treatment.—The milder forms usually recover with self-medication. The severe forms are the ones for which we are more frequently called to prescribe.

General Management.—Having made the diagnosis and after having ruled out the possibility of a peritonitis from any other cause, the first indication is to clean out the gastro-intestinal tract. Please note the expression, *after having ruled out peritonitis,* as it is unquestionably bad practice to promiscuously administer an active cathartic in the presence of acute peritoneal involvement. It is sometimes difficult with a history of tainted foods having been eaten, in an acute abdomen, to determine on a course of treatment. If diarrhea has not already begun and if we are not perfectly sure of our diagnosis, it is safer to wait from two to six hours until the symptoms are more definitely presenting than to promptly administer a purge. A word of warning should be sounded to all the profession that when we are informed that this or that patient has a pain in his abdomen we should not order by telephone the administration of a cathartic. We should see the patient, examine the abdomen, elicit tenderness if present, test for a state of rigidity or relaxation of the abdominal musculature, and, if necessary, have a **blood count** made before we order a purgative.

Having then determined that the patient has an acute inflammation of the gastro-intestinal tract, toxic, infectious, mechanical or otherwise, our first procedure is to **empty the stomach and the bowel.** This is best done with a dose of **castor oil** of from ½ to 2 ounces (15.50 to 62.20 mils) without the preliminary calomel administration. The stomach may be washed out and the colon cleaned by simple **enema** in those patients exhibiting paresis of the bowels with no audible peristalsis. **Repeated flushings of normal salt solution** from a **fountain syringe elevated no higher than two and one-half feet above the level upon which the patient lies** may be given. If the pain be very severe and shocking, and the vomiting and diarrhea are already excessive, temporary relief may have to be given the patient. This is best done by a quarter grain (0.0162 gram) or more of **morphin sulphate** for the adult (never for the child), administered hypodermically. Counter irritation may be applied to the abdomen by the good old-fashioned **mustard plaster** made as follows: Mustard flour 1 tablespoonful, white flour 4 tablespoonfuls, glycerin 1 teaspoonful, white of one egg and enough cool water to make a paste. This is spread between two layers of muslin and applied without previous warming. This is specified because a number of patients will volatilize the oil of mustard by warming the plaster in an oven before applying. This plaster has also been described in some detail for the benefit of those who may not know how a mustard plaster should be made. This plaster may remain on two or three hours and will not blister the skin. It is slow in action and when removed, the hyperemia which it produces is long-lived. **Turpentine stupes** may be used over the abdomen, as well as the **Priessnitz compress** consisting of several layers of flannel wrung out in hot water, covered with oil silk and overlaid with a hot water bag or electrically heated pad. Such preparations as kaolin and glycerin mixtures or the proprietary ones which are on the market, the authors believe are far less useful. In children the **hot foot bath** or **sitz-bath** may be beneficial. One simple but useful procedure that should be spoken of is the immediate administration of **fuller's earth** at the onset of an acute enterocolitis. One ounce (31.10 grams) of this should be mixed with a glassful of water and taken in two doses, one-half hour apart. This is a most unpalatable dose, almost like drinking mud, but can be improved somewhat by flavoring with coffee, chocolate, peppermint or other things. This reaches the intestines as a bolus and due to its absorptive properties literally sponges off the intestinal mucosa and helps to get rid of the inflammatory matters. The authors have seen this act most favorably in certain acute cases. It should be followed a few hours later with a full dose of castor oil.

Having then made the patient temporarily comfortable with some of the above procedures, thereby meeting the immediate conditions requiring attention, it then becomes necessary to prevent further trouble as well as allay the active inflammation which we are combating.

In the first place the **abstinence from food** is of paramount importance; indeed, this alone in many cases will lead patients to recovery, especially the child. As a substitute for food here we may use **barley water** or **flaxseed tea.** The authors dislike the use of albumen water as it may further add to the intoxication through putrefaction of the albumenoid substance. In the adult the best substitute for food is perhaps found in a mild alkaline drink. Fortunately these patients are not hungry and even have a distaste for food, so that we can frequently allow over twenty-four hours to elapse before further feeding is absolutely required.

At the end of this time, if the diarrhea has abated and the appetite is returning, the temperature is lowered and the patient is otherwise symptom-free, it is safe to begin nourishment. In the child perhaps the easiest way to begin is with a low dilution of **malted milk**, or a starchy food as represented by a thin **cream of wheat** or **farina,** for six hours, in small two-hour feedings, and if then there has been no return of symptoms, there may be substituted more solid starchy foods, in all cases giving **preference** to the **starch over sugars, albumens and fats. Mashed potato, strained oat meal gruel, and soft boiled rice** are to be taken for another twenty-four or forty-eight hours while the patient is still resting in bed. At the end of this time, should no symptoms return, the usual diet can gradually be resumed, so that at the end of one week the patient is on a rationally modified whole diet.

MEDICINAL TREATMENT.—During this time it may be necessary to administer some medicines, depending upon the intensity of the presenting symptoms. If the lesion be well up in the gastro-intestinal tract, involving the stomach, duodenum, or jejunum, nausea and vomiting may be the complaints which we will most frequently have to combat. Usually if the previously mentioned treatment has been followed out carefully we will not need much medicine. The simpler the medicine can be made, the sooner the patient will get well. The authors are opposed to the administration of large quantities or numerous kinds of medicine. So often the doctor feels that to satisfy the patient he must write a prescription, when as a matter of fact, in his own heart he knows that simple dietetic measures and proper hygiene will get his patient well faster than his pharmaceutical concoctions.

If nausea and vomiting continue, then we are actually called upon to administer some medicine. An eighth to a quarter of a grain (0.0081 to 0.0162 gram) of **cocain** for an adult, *never for the child,* combined with a little **cerium oxylate,** grains 1 or 2 (0.65 or 0.13 gram), and perhaps **subcarbonate of bismuth,** 15 to 20 grains (1.0 to 1.3 grams), in one or two doses, may allay this annoying symptom. Or we may administer a mild alkaline drink, made up of the following prescription:

℞

Sodium sulphate	ʒi	(4.0 grams)
Sodium phosphate	ʒi	(4.0 grams)
Sodium bicarbonate	℥ii	(62.2 grams)
Calcium carbonate	℥ii	(62.2 grams)

Sig.—1 teaspoonful in half a glass of cool water, to which 10 or 15 drops of the *elixir of catnip* and *fennel* may be added for flavoring, every one or two hours.

This may entirely relieve the symptoms. If the pain has not been relieved, the dietary then requires further modification and curtailment. It is not necessary, as a rule, to use an opiate, but if required the **camphorated tincture of opium,** 10 to 60 drops for one or two doses, at hourly intervals, will usually suffice.

With diarrhea continuing the diet should under no circumstances be increased. This is best controlled, then, by the use of **bismuth subcarbonate or subgallate,** in 10 to 30 grain doses (0.65 to 1.95 gram) every two to three hours, combined with **salol,** 1 or 2 grains (0.065 or 0.13 gm.), and **tincture of kino,** ten to fifteen drops in a dose (for its astringent effect) in **chalk mixture,** and **syrup of ginger,** equal parts.

Of course where marked depletion has taken place the fluids of the body may have to be rapidly replaced, and it may be necessary to practice **hypodermoclysis** if **enteroclysis** is not feasible, or a 25 per cent. glucose in sterile normal salt solution may be given intravenously up to 200 mils. The authors prefer to let the bowel alone as far as topical applications from below are concerned. But in the child the most useful method of allaying a serious colitis is by the **injection of a starch solution** containing from 2 to 5 drops of **laudanum (tincture opii).** This is made up just as a starch for laundry purpose is prepared; one teaspoonful of powdered starch is moistened with cold water and brought to a consistency of a paste. Over this one quart of boiling water is poured and allowed to cool down to about 100° F. (37.22° C.). If this is then too thick to flow through the nozzle of a **fountain syringe** it is thinned still further with water at body temperature. From one-half to one pint, depending upon the size of the child, is run in through the anus by gravity, with the hips elevated and the bottom of the fountain syringe not more than one and one-half feet above the level at which the child lies. This may be repeated every two or four hours until the troublesome mucous discharge is lessened. Rectal prolapse is liable to occur in these cases and should be immediately reduced with the finger well anointed with vaseline.

The authors do not believe that much is ever accomplished by the so-called washing of the acutely inflamed colon, but Graham[12] reports the use of **salt solution** by the **Murphy drip method,** as a helpful measure where marked depletion has occurred.

Prognosis.—The prognosis is usually good. The milder forms frequently progress to recovery without treatment and it is rare for them to extend beyond a week. The more severe types usually require medical aid. With proper treatment and modification of the diet, recovery takes place, as a rule, within from one to four weeks. In the case of the follicular enterocolitis of children, recovery perhaps is not the rule, as this form frequently passes into the chronic types, even under the best of treatment.

Pathology.—ACUTE CATARRHAL COLITIS.—In this condition one finds increased mucus coating the surface, mixed with exfoliated degenerated epithelium and red blood-cells. The mucous membrane may show only moderate congestion, or, in slightly more advanced states such as ptomain poisoning, there may be serum and fibrin and leukocytic infiltration of the submucosa and purulent exudate. The mucous cells may be distended, giving a granular appearance to the surface. This thickened mucosa is sometimes edematous from serum transudation and may become secondarily infected by intestinal bacteria leading to destructive lesions.[13] Where the process has been of longer duration and scar tissue begins to be deposited within the mucosa between the glands and in the submucosa, the natural mucous glands become crowded and the whole structure thinned. This is atrophic catarrh and is essentially a chronic type and finally small but widely disseminated ulceration may occur.

The exudative or suppurative type [13] is rare. This is due to embolism resulting in obstruction and strangulation of the blood supply of segments of the bowel by purulent material.

ACUTE INFECTIOUS COLITIS (TROPICAL DYSENTERY).—(*a*) Amebæ coli, protozoa, early essentially exudative, later, hypertrophic and still later ulcerative. The same may be said for: (*b*) Bacterial, mainly bacillary, Bacillus coli, Bacillus Shiga, Bacillus Flexner, Bacillus Morgani, Bacillus pyocyaneus, and Streptococcus pyogenes. (Refer to chapters on Dysentery, pp. 271 to 358.

FOLLICULAR OR NODULAR TYPE.—There is epithelial exfoliation with leukocytes and mucus in the early stage, presenting a reddened coarsely granular surface with punctate nodules studded over the surface. These are the solitary follicles, and range in size from a pinhead to a split pea.[14] After the first week enlarged follicles, or lymph-nodes, and Peyer's patches, cellular hyperplasia with erosions, and ulcerations, or a diffuse round cell infiltration may occur causing thickening of bowel wall. This may be accompanied by a croupous colitis in which a false membrane (coagulation necrosis) will be seen in splotches and may be firmly attached by fibrin ramifications extending through the mucosa, together with pus, as far down as the peritoneal covering. This may proceed to necrosis of only the surface membrane or suppuration and ulceration may involve the deeper coats. This was the type leading to most of the fatal cases in the study made by Zahorsky.[15]

OTHER TYPES.—In *necrotic colitis* the submucosa is mainly attacked, and the mucosa is undermined by infecting organisms mainly of the pus-forming variety leaving ulcers with freely overhanging edges. This is rapidly fatal.[13]

A subacute variety which really belongs to the chronic type of colitis is the *mucomembranous colitis*. This may be inflammatory in nature but it has as its chief feature an outpouring of the contents of the mucous glands, so that there is an excessive mucus formation with the coagulation of mucin. When to this there is added an excessive desquamation of the surface epithelium, shreds and skins, at times shaped to the various convolutions of the bowel, are passed. Fibrin does not seem to be included in the structure of these mucous sheets.

In anthrax infection (mycosis), Bacillus anthracis is the cause. It is usually found in the small intestine, and is characterized by a studding of the mucosa with small brown and black elevations, with areas of hemorrhage and edema or ulcerations from which the anthrax bacillus can be recovered.[18]

CHRONIC ENTEROCOLITIS

Synonyms.—Conditions synonymous with chronic enterocolitis are: Chronic catarrh of the intestines; chronic ileocolitis; chronic catarrhal enteritis; chronic diarrhea; mucous colitis; mucomembranous colitis; ulcerative colitis; chronic intestinal indigestion; dysentery; atrophic and hypertrophic enterocolitis.

Definition.—Chronic enterocolitis is an inflammation of the small and large bowel, with or without ulceration, which persists for more than four or five weeks after its acute inception. Such an enterocolitis may range from the mildest type of chronic intestinal indigestion up to ulceration and even to perforation.

Etiology.—PREDISPOSING CAUSES.—All the factors outlined under the etiology of the acute stage are to be considered in producing or maintaining a chronic enterocolitis. Once having developed an acute enteritis, a lack of care in dietary during its convalescence or an improper management of the acute infection may produce a state of chronic enterocolitis. Perhaps the most important factors are the too early feeding and lack of rest during and following an acute attack. Failure to recognize the factors contributing to the initial attack is an extremely important etiologic point. If, for instance, we have exposure to cold, improper manner of eating and incorrect dietary and if these factors are not recognized and corrected, a chronic process is very apt to result. Many individuals suffering with this disease are not in balance with their surroundings. There may be a necessity of their dining at cheap restaurants or poor boarding houses; the climate of the city in which they live may not be satisfactory for them; long hours of uncongenial work or too much social activity may militate against them. The investigation and correction of these points are necessary. The authors believe that heredity plays a very important part in the production of the atrophic type of enterocolitis. An individual may inherit as one of the parental physical characteristics a weakened and nonresistant gastric mucosa. The authors believe that a familial type of achylia gastrica is possible, although not yet proved and they believe that the same process might occur in the enteric mucosa. It is perfectly reasonable to suppose that a group of related individuals may have a poorly resistent mucosa that suffers with the slightest traumatism which would affect others not at all. The authors have never seen anything published in regard to this hypothesis, but they believe that such a supposition is entirely tenable.

Among the toxic agents, long-continued chronic alcoholism, the use of mercurial and arsenical preparations, and perhaps the excessive use of sodium chlorid and condiments may be contributing and even exciting causes. As in infections elsewhere in the gastro-intestinal tract, infections in the tonsils, sinuses, teeth, gums and bronchial tree play a very important part in keeping alive chronic infection in the small and large bowel. Malformations such as diverticuli, Jackson's membranes, Lane's kinks, and postoperative adhesions, which may interfere with the normal enteric current, may be responsible for maintaining a chronic infection. In like manner, tumors partially obstructing the bowel, displacements of pelvic organs, and inflammatory infiltration of the left uterosacral ligament, may interfere with the normal function of defecation. Anal conditions such as fissure and hemorrhoids may be responsible for impairment of normal defecation. Parasites, particularly the flagellates, by traumatizing the bowel wall and by toxin elaboration may possibly be an exciting cause.

Symptomatology (Not Including Ulcerations).—Chronic enterocolitis as a continuance of those acute types, mainly of milder character, extends over a period of four or five weeks to many years.

General Consideration.—These patients represent many types of the chronic intestinal invalid. They may show little external evidence of the condition or they may be wasted and thin. They are frequently mentally submerged and at odds with the world. Their history is obtained with difficulty; they do not remember, or it is too much trouble to recall facts and dates. They feel generally miserable or vaguely "mean" and this may manifest itself as an internal discomfort which may be only a fullness or bloating of the abdomen when the small bowel alone is involved; they may have borborygmus and flatus passage, more foul when there is much proteid decomposition. Their headache is usually dull and diffuse, but often occipital. There may be backache and lassitude. They lack stamina and are easily upset if they do not take the best care of themselves. The symptoms vary from those of a mild intestinal toxemia to the severely toxic states. The patients are frequently ptotic and their habitus is bad. They stand with drooped shoulders and protruding abdomen and slouch when sitting. The complexion is muddy or swarthy, the eyes dull and the tongue may be flabby, thick, tooth-marked and coated and there is frequently a slight cyanosis of tongue and possibly of lips. Anorexia or erratic appetite is present with other evidences of hepatic dysfunction from long-continued absorption from the large bowel of improperly digested foods and abnormal gases (hydrogen sulphid) [16] which are carried by the portal circulation into a long-suffering liver. They may or may not have diarrhea—usually they do have it if the colon be much involved. It may manifest itself by one to four loose movements a day. These may begin upon awakening and all may occur within the first one or two hours after arising, with no further bowel movements during the day. This type frequently is seen associated with achylia gastrica. Or the bowel movements may be scattered regularly throughout the day and night, some patients being required to arise once during the night to defecate. Other patients will have one formed movement on arising, shortly followed by several loose ones, often accompanied by violent explosions of gas, or again the first movement (in sigmoid and rectal involvement) may be mucoid and then shortly thereafter fecal or mucofecal. Where the small bowel alone is involved or possibly with it the cecum, there may be only infrequent diarrhea, or none at all.

The feces in this type is sticky and slimy, adhering to the toilet vessel, and has a smooth mirror-like surface, is semiformed and rarely mushy as when the involvement is farther down. Grossly, digestion is fairly complete but by microscopical examination are found undigested food elements such as meat fibers, usually unstriated, where proteid digestion has failed, or large amounts of starch in carbohydrate indigestion, or free fats and fatty acids in failure of fat conversion. This is commonly thought to be suggestive of pancreatic and hepatic dysfunction, but may be due in part to the abnormally hurried intestinal motility, so that sufficient time is not allowed for proper enzymatic action.

Or constipation may be complained of, and when present is usually of the atonic type. Atony exists in that part of the large bowel where the stool begins to take form, namely the splenic portion of the transverse colon, the descending colon and sigmoid, and the stool is apt to be large in the diameter of its transverse section, smooth and glistening, and the patient complains of lack of power to expel it. Or spasticity rather than atony of the descending and pelvic colon may also produce constipation. Frequently the descending colon and sigmoid are found to be spastic by palpation, and they can be rolled under the fingers. By roentgen ray it shows as a smooth tube without haustra and in a sigmoidoscopic study the introduction of the scope will be difficult past O'Byrne's sphincter (rectosigmoid juncture), and beyond this point the sigmoid will often be tightly contracted around the advancing end of the scope and will not balloon out by normal atmospheric pressure, and each inch of advancement will have to be accomplished by air dilatation. Here there is no intestinal lack of power but rather a blocking of the fecal pathway by hypermuscular activity. When the spasm is overcome the contents are expelled in one wild rush, usually with explosive flatus discharge, and on examination the stool is found to be of very small caliber, pencil size, perhaps twelve or fifteen inches long, and may show free and intimately mixed mucus. With spasticity above and atony below, hypersegmentation is evidenced by a conglomerate fecal movement of closely packed small masses.

MUCOMEMBRANOUS COLITIS.—While it is a moot point whether this be an actual inflammatory condition, there are reasons for believing that inflammation, perhaps of a very low grade and of chemical causation, is the underlying etiological factor of this condition. A careful discussion of this subject is taken up in another chapter so that we shall only allude to it here.

The patient is usually a frail, neurotic and ptotic woman; toxemia is evidenced by the mental state which may be irritable, introspective, suspicious, excitable, or depressed, fault-finding, frequently hysterical, or the patient may even be found bordering on insanity. Added to this the skin is frequently swarthy and there may be a history of eczema, urticaria, asthma, angioneurotic edema and erythema multiforme.

Constipation is often of many years' standing and of the atonic type. These patients are essentially atonics in the whole gastro-intestinal tube. Intestinal stasis with abnormal proteolysis and histamin formation[17, 18] probably precedes the actual condition for a long time. As the condition progresses, attacks of diffuse colicky pain or "queer" discomfort are frequently added, to be followed by the prompt expulsion of hard feces bearing longitudinal bands of thick, tenacious mucus and possibly sheets of mucus or even casts of the bowel. These on analysis seem to be coagulated mucin and do not show the fibrin so often present in inflammatory exudates. It has been shown by Rogers[19] that a ferment mucinase will coagulate mucin, thus explaining the formation of the membrane.[20] The bile contains an antimucinase which destroys the mucinase and therefore Rogers suggested the use of ox gall in treatment. Attacks of paroxysmal diarrhea may be added and are probably the result of a localized acute colitis set up by secondary infection. The descending colon may be found spastic and the ascending atonic. The sigmoidoscope discloses a reddened mucosa, in some cases, with mucous bands which often have a dead-fish-like odor.

Zugsmith[21] in an excellent paper on this subject points out that too often patients are operated on for a so-called chronic appendicitis without any permanent improvement. The appendix, while it may have been somewhat at fault, was not the chief malefactor. It was only showing signs that were present in far more extensive proportions in the cecum, of which it was a part. So, too, the gall-bladder is frequently sacrificed when it is sometimes only a relatively inconspicuous element in a widely disseminated process of inflammation involving the bowel and the bile passages.

Diagnosis.—The diagnosis of chronic enteritis is not always an easy one. One has to rule out chronic enteric toxemia, if one cares to split the diagnosis to its finest point. The differentiation between the two is not easy nor really of very great importance. It is hard to tell where a chronic intoxication leaves off and enterocolitis begins. In fact some authors believe that all intoxicating processes are essentially inflammatory. Whether the bowel wall or the bowel content be infected is a nice point in diagnosis. In general, however, the authors think that the finding of mucus, either free or incorporated, is the dominating element in arriving at a diagnosis of an inflammatory process in the bowel. Here again, as in the acute conditions, the segment of bowel involved will best be located by studying the stools, and you are referred to this subject under acute enterocolitis. Uncomplicated ptosis has to be differentiated from this condition, and here again the stools and motility tests (see Foreword) are of most value.

In ptosis we are apt to have a greater motor delay than in the inflammatory states, but this is not always so.

Surgical intestinal conditions, in which the lesion is obstructive, are best ruled out by x-ray.

Bright's disease, pulmonary tuberculosis and hepatic cirrhosis, as we have pointed out before, may all have an associated enterocolitis which obscures the true underlying condition.

Malignancy involving the various intra-abdominal organs must always be thought of in arriving at a proper diagnosis. This diagnosis is extremely difficult to make in its early stages and indeed is frequently impossible until a palpable mass is found. And here the diagnosis comes usually too late. Every case of enterocolitis coming under observation should suggest in an adult in the neighborhood of the early forties the possibilities of a coincident malignant involvement. Here x-ray and possibly laparotomy should be resorted to in making a diagnosis.

Gastric and duodenal ulcer are frequently mistaken for a chronic enterocolitis. In both conditions one may get a history of regular pain at a definite time after eating, because of the fact that eating excites intestinal peristalsis which will produce pain in enterocolitis. Here gastric and duodenal studies will frequently set us right and allow us to rule out ulcer. Of course, a coincident ulcer may exist with enterocolitis.

Gall-bladder infections may give us symptoms similar to those of enterocolitis, but here again, as in ulcer, our chief reliance is placed on examination made by the duodenal tube in practicing such non-surgical biliary drainage as Lyon has advocated.[22, 23]

The only evidence we may have of a duodenitis, except for some vague gastro-intestinal complaints, will be found in the study of the duodenal fluid extracted by means of the duodenal tube. In studying gall-bladder cases, in which a routine examination is made of the duodenal fluids preparatory to the drainage, the authors have been surprised to observe the number of patients having an associated duodenitis. This diagnosis is made on the nature of the return of the duodenal washings just as evidences of gastritis are found in the stomach washings. Frequently the return fluid will be quite viscid and contain a number of floccules which on microscopical examination may show many exfoliated duodenal cells, oval or cuboidal in shape and somewhat larger than a white blood corpuscle. Together with this will be seen many white blood-cells, sometimes red blood-cells, and thick mucous strands with myriads of bacteria, streptococci, staphylococci, groups of bacilli, and even colonies similar to those seen growing on the surface of a Petri culture plate. Culturation will frequently grow out streptococci, staphylococci, Bacillus coli and many other organisms. This work of duodenal intubation has frequently led the authors to the diagnosis of Giardia or Lamblia Intestinalis infection as an exciting cause for duodenitis, whereas this condition might readily have been overlooked if they had relied on other measures alone.[24]

SIGMOIDOSCOPY.—Too frequently the lower end of the digestive canal is neglected because of the difficulty of making an examination. The old sigmoidoscopes of large caliber too frequently produced pain in their introduction. Recently the authors have used, at Dr. Soper's suggestion, a scope of five-eighths inch diameter. This is a full quarter-inch below the size of the Tuttle and Lynch scopes which they formerly were in the habit of using. The authors have modified the lighting of this instrument so that, instead of the distal end of the scope being illuminated, they have adapted the Lynch type of lamp operating at the eye end of the scope. This allows them now to make examinations that were frequently denied them. By the use of this instrument various stages of inflammatory changes can be noted in the mucous membrane, and the tonus of the rectum and sigmoid can be very efficiently estimated by inspection. The completeness of defecation is readily observed. The response of the mucous membrane to irritation by enema is seen by the outpouring of mucus in the sigmoid and rectum immediately after its use. If the authors find the tone and the mucous membrane of the rectum and sigmoid normal, but encounter sticky feces covering the walls of both these structures, they believe that this is their best method of diagnosing an enteritis or an enterocolitis that is not involving the lower bowel. Frequently one encounters by this examination normal feces lying in an inflamed indurated or atrophic rectosigmoid. In these cases one feels justified in making a diagnosis of lower colon infection, believing the upper small bowel to be uninvolved. An ileocolitis of the atrophic type may show a very crumbly and shaggy stool from lack of lubrication in the transverse and descending colon. The mucous membrane will appear dry, glistening, polished and shrunken so that the vessels stand out prominently. The mucous membrane will tend to pile up easily on the end of the scope as it is introduced and is decidedly thin. Associated with this one will usually observe a lack of tone in the musculature. Ileocolitis of the hypertrophic type exhibits a thickened, engorged, moist, and a frequently pigmented (hemochromatosis) mucous membrane, in which only a few vessels can be recognized. The feces may be thin, and the mucus quite abundant. This type is likely to be hypertonic, the valves of the rectum are thickened and stiff and the wall of the bowel seems indurated. Difficulty may be met with in passing beyond the rectosigmoid junction and because of the stiffness of the bowel wall it is frequently difficult to introduce the scope very far into the sigmoid flexure.

Treatment.—GENERAL MEASURES.—A proper **diet**[25] is of paramount importance and, in general, should include a **minimum of proteid and a maximum of carbohydrates,** with especial care as to the **avoidance** of **excesses of sugars** and **excesses of cold or iced drinks.** The **vegetables** should be **finely divided** by passage through a **colander** which will take out the hulls of beans and peas. **Potatoes** should be **well baked** or **mashed.** Rough vegetables such as cold-slaw, improperly cooked cabbage or cauliflower should be avoided. When **meat** is taken it should be put through a **meat grinder** and should be thoroughly chewed. Fried, rich and greasy foods and rich pastries must be avoided. Condiments often act as poisons in these cases. Stewed fruits, if free of fibers, may be taken with care. As a rule, raw acid fruits, and berries are not well borne. As a check-up on the diet, a frequent microscopic examination of the stool is perhaps our best means of determining the relative amounts of starches, fats and proteids that should be administered. Insist on thorough chewing of foods.

The various factors which have led to a continuation of this condition as mentioned under etiology, relative to the patient's surroundings, type of work, climate, and methods of feeding should be rectified. These patients require **plenty of rest**, especially after meals, as motion increases peristalsis and this in turn is bad for the inflamed bowel. They should particularly be protected from undue exposure in changes in temperature, and some of these patients experience a great deal of relief in wearing a **flannel band** around the **abdomen.** Where ptosis is a dominant feature, the proper abdominal support with a **Curtis plate** or a **special corset,** of which the **Gossard** is a very good type, should be applied. **Plenty of water intake** should be the rule; yet a great many of these patients complain that they are unable to drink water without discomfort. Here the temperature may have to be modified as the spastic type of colitis will not take cool water happily, whereas it does very much better when hot drinks are administered. The reverse is seen in the atonic cases. Many of these patients will as a rule take **flaxseed tea** with comfort where water is not well borne. This is prepared by pouring a pint of boiling water over a tablespoonful of the whole flaxseed and allowing it to steep on the back of the stove for four or five hours. A little sugar may be added, or lemon juice, to taste.

Exercise and **massage** are beneficial at times but massage of the abdomen must be practiced with the greatest of care and lightness of application; otherwise damage may be done to the inflamed bowel. The authors find in their atonic cases that the best form of massage is by way of the electric current as applied by the **Morse sine-wave machine.** This is gentle and helpful.

MEDICAL TREATMENT.—Laxatives in all cases are harmful. For years the authors have been laying stress on the importance of avoiding laxatives.[7] A great many of these constipated cases have been subject to the laxative habit for years and it is a difficult thing to break this habit; yet it must be broken before the patient will recover. The authors have found the use of a fruit mixture such as the following, very useful:

Figs	½ lb.
Dates	½ lb.
Prunes	6 or 12, stewed with their juice.

This is to be run through a meat grinder and the following mixture is added:

Powdered senna ½ ounce (15.50 grams)
Granular agar-agar ½ to 1½ ounces (15.50 to 46.60 grams)

The whole is patted into a paste and kept in the refrigerator. The dose is begun with a piece the size of the end of the thumb with or after each meal, and the quantity gradually reduced as the bowels respond until eventually only a half portion is taken after the evening meal; then the whole formula is remade, *omitting* the senna, and the dosage is repeated as with the former mixture, again gradually reducing the amount until the bowels are responding at normal intervals. Of course during all this period it is necessary to insist that the patient have a definite time for going to stool each day.

The administration of **bile salts**, in any of the well-known **pill forms** on the market, either alone or combined with **pancreatic extracts**, one or two hours after meals, is a very useful procedure, for its effect on fat assimilation, as a cholagogue and for its antimucinase action. **Mineral oil** is useful in some cases but the authors prefer to get along without it wherever possible, as many of these patients are poorly nourished primarily, and the coating of the undigested food particles with a film of viscid oil, they believe, must prevent some digestion and assimilation. Therefore where they do use it, they instruct that it be taken only on retiring, thus allowing most of the digesting food to precede the oil in its transit through the small bowel. As lubrication is needed only in the lower large bowel it will thus have ample time to overtake the bowel contents and be thoroughly incorporated with it and aid in its passage.

Nerve sedatives, such as **bromids**, **sumbul**, **asafetida**, and **valerian** are often indicated and form a very important group of useful drugs in the treatment of this condition. **Tincture of belladonna**, as well as the **fluid extract of drosera**, which may be combined with it and increased up to the physiological effect, are frequently very useful in overcoming chronic spastic states. The authors cannot say the same for benzyl benzoate. While this drug may be extremely useful in acute spasm of unstriped muscle, in their experience the cases have been few that have been benefited by its prolonged use.

Soper [26] has suggested to the authors the rectosigmoidal instillation of from 1 to 3 ounces (31.10 to 93.30 grams) of a 25 per cent. (a half saturated) solution of **magnesium sulphate** in the spastic type. In the atonic type, to this solution may be added an equal volume of 25 per cent. **sodium sulphate solution.** A prompt bowel action follows within fifteen minutes. The authors have found this to be a useful procedure, while withdrawing the use of laxatives, to aid in establishing a habit

With patients who have diarrhea rather than constipation the authors feel that this is best controlled by modifying the diet, which will often be all that is required. If diarrhea is persistent, then some medicine will have to be administered. Frequently the use of 2 to 3 grains (0.13 to 0.195 gram) of **ichthyol** two hours after meals in capsule or pill will be sufficient to allay irritation and quiet an excessively acting bowel. Likewise **bismuth** in 10 to 15 grain (0.65 to 0.972 gram) doses may at times have to be added.

Topical treatment to the small bowels such as that proposed by Jutte,[27] Palefski,[28] Einhorn [29] and Buckstein [30] may be helpful. In all of these methods the principle is much the same. It consists in passing a **duodenal** or an **intestinal tube through the fasting stomach into the small bowel.** The tube is allowed to pass to any desired length, depending upon the portion of bowel that it is desired to treat. Simple washing of the bowel with plain water, hypertonic, or tonic salt solutions and even the injection of **oxygen gas** have been claimed to be beneficial. The endeavor here is to remove in a mechanical way as much of the infected intestinal content as is possible by the mechanical flushing, and to apply topical medicaments. The oxygen is used for its effect on the anaërobic organisms present.

The authors are not strong advocates of chronic flushings, as they have felt that many times the patient's condition has been kept active by irritating solutions thrown into the bowel. Those flushings which are most frequently used are: **silver nitrate,** from 1 to 2 grains (0.065 to 0.13 gram) in a quart of water; **copper sulphate,** 2 to 4 grains (0.13 to 0.26 gram) to the quart; **mercurochrome,** in one-half to one per cent. solution; and **plain lime water.** Simple washing of the bowel with **normal salt solution** is sometimes indicated. Stauffer [31] administers a 10 per cent. solution of **eucalyptol** in **olive oil** and has it retained for two hours. In patients having hard scybalous movements, such as are frequently seen in mucous colitis with mucous bands and casts, it may be necessary to remove the scybala by colonic flushings with a 2 per cent. **hydrogen peroxid solution.**

Following Soper's [32] suggestion, the authors have found that the cases showing involvement of the rectum and sigmoid have been materially benefited by the insufflation of a **calomel** and **bismuth powder** in equal parts. The small scope is introduced as far into the sigmoid as one cares to go and, with a long nozzled powder blower, the powder is applied directly to the mucosa as the scope is withdrawn. This treatment is given every day for one or two weeks and then every other day for a week or two and thereafter less frequently. Even where the bowel is involved higher than can be reached with the scope, the authors have seen improvement in a number of cases follow this method. It is only necessary to refer to the importance of **correction of hemorrhoids and fissure** as a preliminary to the beginning of treatment.

Bassler [16] reports brilliant results following the use of **antagonistic bacterial cultures introduced** directly into the **colon** by the **catheter.** Besides this he modifies the diet according to the nature of the type of infection which he finds. Together with this form of treatment **autogenous vaccines** are sometimes prepared and administered hypodermatically. The use of bacillary tablets and cultures has been advocated for a long time, perhaps foolishly, for it has been shown by Kendall,[33] Rettger,[34] Norman [35] and others that the Bacillus bulgaricus cannot be permanently transplanted to and made to thrive in the human intestine. Recently the authors have seen a method of **direct bacterial instillation** described by Norman.[35] Here the colon is first thoroughly washed out with salt solution to cleanse the bowel and a living culture of **bacillus acidophilus** is injected. This method appeals to them as being rational in the indolic types of infection. Together with this, **milk sugar** is added to augment the pabulum on which these bacteria must subsist.

The authors have further modified this method of treatment by administering, through the duodenal tube, directly into the jejunum, 30 mils of a broth culture of the bacillus acidophilus, incubated for sixty hours. This is added to 60 mils of sterile water in which 1 tablespoonful of lactose has been dissolved and allowed to run into the intestine at a temperature of 100° F. (37.7° C.). Following this from 225 to 500 mils of the watery-lactose-culture of bacillus acidophilus, incubated for twenty-four hours, are instilled by catheter into the rectum or sigmoid.

Prognosis.—The prognosis in chronic enterocolitis cannot be foretold in any individual patient until his response to treatment has been learned. On the whole, the authors believe, however, that the prognosis in this condition is more favorable than has been generally given. The method of treatment used must be individualized with each case and several elements, or in certain cases, all of the measures of treatment suggested previously must be used in their care. Many of them appear at first to be hopeless intestinal defectives and border on the chronic intestinal invalid, but even in such cases by persistence and patience and the adoption of certain of the methods advocated by Bryant,[36] unexpected symptomatic recoveries will be accomplished.

Pathology.—MORBID ANATOMY.—The morbid anatomy of chronic enterocolitis is such as was seen in acute catarrh with hyperemia of the mucosa and with mucus bathing the surface, but differs from the acute in that it is not transient but permanent. This may be limited to the villi which may be found swollen or hypertrophied (polypoid), or atrophied, or the mucosa may show a bloody extravasation and pigmentation. In the milder catarrhal forms the mucosa alone suffers by a disappearance of the tubular glands with lymphoid hyperplasia. If the process be of longer duration or more severe, the submucosa and muscular coat may show evidence of thickening from edema, serous fluid infiltration or round cell proliferation finally eventuating in connective tissue formation, a characteristic of the chronic stage in which the bowel wall may be two or three times the usual thickness. The mucosa may be atrophied or shrunken and indeed the muscularis may have lost its tone and allow the bowel to become dilated and atonic.

In the follicular type, more common in children, the process has advanced from the stage of lymphoid tissue hyperplasia and cellular proliferation to necrosis of the mucosa overlying the nodules with degeneration of the nodules, to the formation of small punctate ulcerations with finely granular bases sharply outlined by areas of hyperemia with little tendency to heal.

Where larger ulcers have formed, usually tuberculous, there may be healing with cicatricial contractions resulting in stenosis of the bowel. Mucopurulent discharge is present and the mucous membrane is frequently gray or slate-colored with superficial erosions. The Peyer's patches are pigmented and swollen and the mesenteric lymph-nodes are enlarged.[18]

Multiple cyst formation in the submucosa of the colon has been seen and is due to the connective tissue proliferation, occluding the orifices of the mucous follicles.

Amebic infection is discussed under ulcerations.

The terminal stage of croupous colitis, which is essentially an acute process, may show ulcerations where the necrosed mucosa and submucosa have sloughed away. These ulcers may appear like granulation tissue or they may cicatrize.

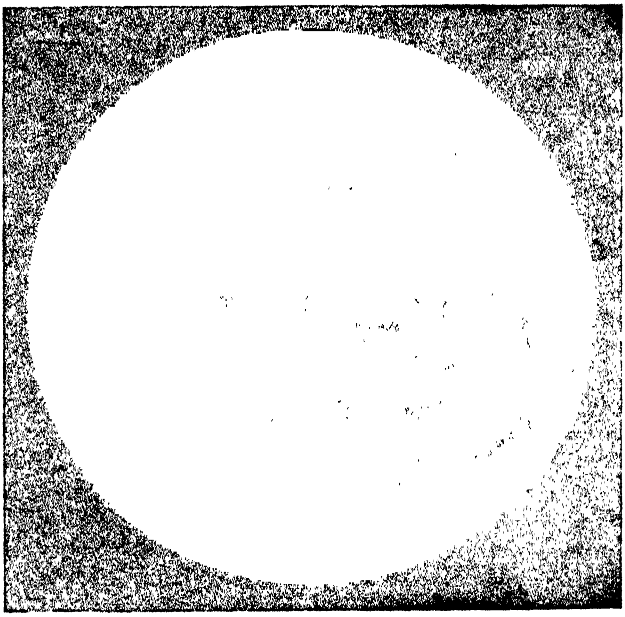

FIG. 1.—PIGMENTATION OF COLON. PIGMENT FOUND IN MONONUCLEAR CELLS OF MUCOSA. (*Dr. H. A. Cotton, Trenton, N. J.*)

In tuberculosis,[18] Peyer's patches and the lymph-nodes are involved and lead frequently to ulcerations. Miliary tubercles may be seen undergoing caseation and the subperitoneal lymphatics appear as white nodular branching cords, extending around the outside of the bowel.

Mucomembranous colitis is described under acute enterocolitis.

Through the courtesy of Dr. Henry A. Cotton,[40] of the New Jersey State Hospital, the authors are permitted to give the following summary of the principal gross and microscopic lesions of the colon and ileum in chronic infections of the intestinal tract. This study was participated in by Doctors Cotton, John W. Draper, John F. Anderson and James Ewing, and represents material grossly and microscopically examined from two hundred specimens removed by resection chiefly from mentally defective or insane patients. Since this study represents the latest and most scientifically conducted work, and, furthermore, since it is concerned with a study of the living rather than dead-house pathology, the authors consider it the most authoritative work upon this subject and think it should be given space in this chapter.

"In the gross specimens the lesions vary in number and extent. In specimens where the infection is of more recent origin, folliculitis is the most common finding. This is especially true of the younger patients. Very marked pigmentation is a common finding. It may be limited to distinct areas, segmental in character, or pigmentation may involve the entire ascending, transverse and descending colon. Congestion of the mucosa is also frequently noted. There is also hypertrophy of the ileocecal sphincter. Very frequently adhesions causing deformities and narrowing of the lumen of the colon are noted. Atrophy is seen in early cases involving the mucosa, while in the more advanced types there is a marked thinning of the muscular coats as well. The rugæ are diminished and often absent through large areas. Diverticulitis is common. Multiple ulcerations, many of which show hemorrhagic centers, are found. In the older type of ulceration there is present an indurated edge with dimple-like center. In the more advanced types of infection the wall of the colon becomes thin. There is ballooning of this portion with constricted areas. Frequently sub-

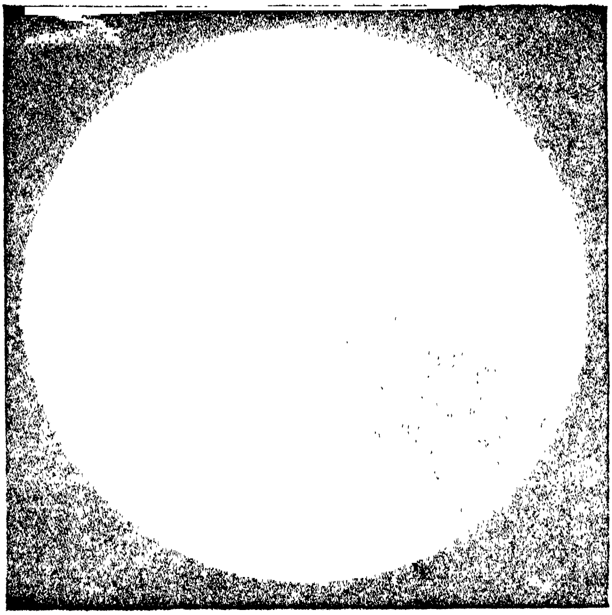

FIG. 2.—DEGENERATION OF MUCOSA (COLLOID). CASE OF MULTIPLE SCLEROSIS. (*Dr. H. A. Cotton, Trenton, N. J.*)

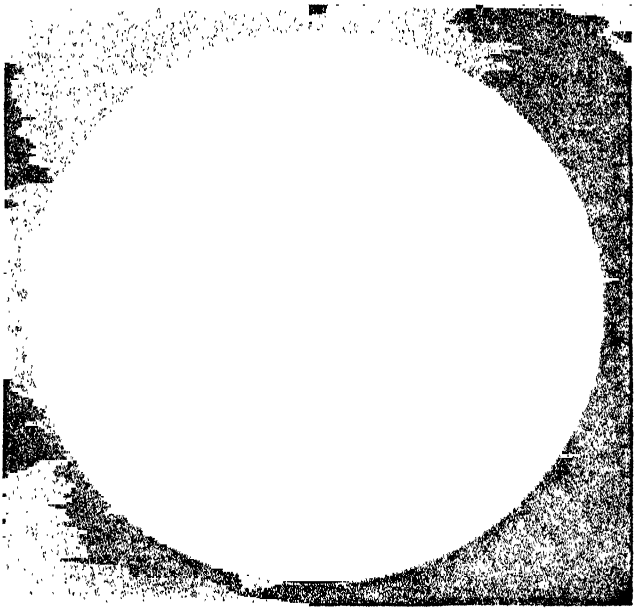

FIG. 3.—HYPERPLASTIC LYMPH NODULE, ROUND CELL INFILTRATION OF MUCOSA AND SUBMUCOSA. (*Dr. H. A. Cotton, Trenton, N. J.*)

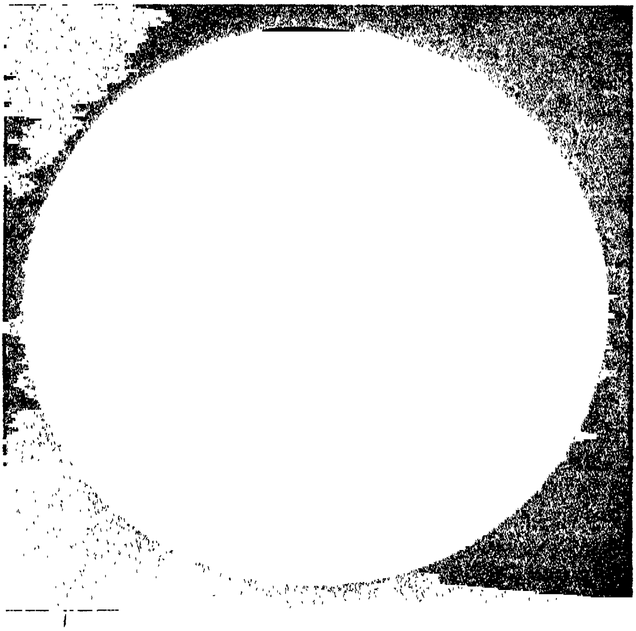

FIG. 4.—FOLLICULAR COLITIS, HYPERPLASTIC LYMPH NODULE AND COMMENCING NECROSIS OF MUCOSA. (*Dr. H. A. Cotton, Trenton, N. J.*)

mucosal hemorrhages may be noted. In some cases a round punched-out ulcer, the size of a dime, with the mucosa broken around the entire edge, is found. One sees frequently a thickening of the sigmoidal walls. The ulceration is most frequent in the cecum around the ileocecal sphincter and appendix. Frequently the ileum is distended, due to the regurgitation of contents through the patent ileocecal valve. In the ileum are frequently found enlarged lymph-nodes which are considerably raised and stand away from the wall. This is a type of nodular hyperplasia. Cicatrization of terminal ileum, and hypertrophy of the ileocecal sphincter are not uncommon. Occasionally the walls of the ileum become atrophied with diminished rugæ.

"One of the most noticeable findings at operation has been the enlarged mesenteric glands, numerous along the ileocecal vessels and in many cases found throughout the mesentery of both colon and ileum and in some cases throughout the whole small intestinal tract. Cultures from these enlarged glands frequently show various types of streptococci and almost constantly various types of colon bacilli. These organisms have undoubtedly found their way through the intestinal wall because of the damaged mucosa.

"MICROSCOPICAL FINDINGS.—In certain areas there is found a marked atrophy of the mucosa, atrophy of muscular coats, edema, round cell infiltration of mucosa, submucosa and tunica propria, with hyperplasia of lymph-nodes. In other types there is marked hyperemia of lymph-nodes with destruction of the lymph glands of the mucosa, pigmentation of endothelial cells, and general hyperemia and engorgement of blood-vessels. It is not unusual to find hemorrhagic areas. In areas with more pronounced destruction there are many young connective tissue cells. Necrosis or flattening of epithelium is frequently noted with ulceration and necrosis of mucosa.

"In the enlarged mesenteric glands the most frequent change noted is hyperplasia. In some there is an endothelial hyperplasia; in others marked degeneration with masses of colloid-like material in the gland, with hemorrhage into the lymph spaces.

"In staining the tissue for bacteria there have been noted Gram positive bacilli and diplococci. Also Gram negative bacilli and cocci have been found in the lumen of the gut, in the tunica propria, and in the hyperplastic lymph-nodules. In many cases these organisms were found to have penetrated the submucosa."[40]

ULCERATION OF THE SMALL AND LARGE BOWEL

Definition.—Ulceration of the bowel is a local necrosis of the mucosa, frequently also involving the submucosa, muscular coat, and visceral peritoneum, and may result in perforation with general or local peritonitis with abscess or adhesion formation. Ulcerations vary in size from pin-point erosions to the diameter of a silver dollar or larger, and may be single or multiple to the extent of a score or more. They may be acute in type, superficial or deep, healing in a few weeks, or chronic, in which case the condition may extend over a period of years.

Etiology.—For the predisposing factors concerned in the production of these lesions of the bowel see section on Duodenal Ulcer. The active causes will be grouped together with special symptomatology and pathology, under the description of each individual type of ulceration which immediately follows the General Symptomatology.

Symptomatology.—GENERAL CONDITIONS.—The symptoms vary from those of an acute enterocolitis to those of a chronic form. This latter is the most usual group observed. Therefore any of the various symptoms previously described under these two forms may be present in this condition. Indeed, it is difficult at times to differentiate between ulcerative and nonulcerative enterocolitis in a patient complaining of colicky pain, diarrhea or constipation and fever. Certain types of ulceration have special symptoms, which will be described below, but in general the marked and continued presence of blood in the stool either macroscopic, microscopic, or occult (the test for which is given on page 485), together with the repeated finding of pus, is indicative of ulceration. The pus will be seen as small green or gray masses, sometimes difficult to find and not constantly present in the mild forms, but if it be large in quantity, in thimbleful amounts, it suggests the discharge of the contents of an abscess. If with this there be certain well-defined and constant points of tenderness over certain areas of the bowel, and if these points be frequently associated with muscular rigidity, the diagnosis is strengthened. Sigmoidoscopic inspection will disclose ulceration of the wall of the rectum and sigmoid when present. The duodenal tube will frequently allow one to make a diagnosis of duodenal or jejunal ulcer by the finding of blood, pus, bacteria and inflammatory débris in the extracted specimens. The presence of mucus and mucous bands is only indicative of inflammation. Bacteria of any type, including tubercle bacilli which are possibly swallowed with the sputum in a pulmonary case, may be found in the feces and mean nothing from the standpoint of ulcer diagnosis except certain specific ones as Bacillus dysenteriæ, Bacillus choleræ, Bacillus lepræ, Bacillus typhosus, or the ameba of dysentery or the yeast fungus of sprue. Fever is a variable factor but is usually present as anemia advances. Diarrhea is more frequently seen than constipation. Pain and tenderness are nearly always present. Emaciation may not develop for a long time unless a larger vessel be eroded, allowing copious and frequent, frank hemorrhages.

SPECIAL TYPES OF ULCERATION.—*Duodenal Ulcer.*—Inasmuch as this subject is fully discussed in another section of TICE, it has not been deemed advisable to again touch upon it here.

Jejunal, Gastrojejunal and Marginal Ulcerations.—Primary jejunal ulceration is unquestionably a rare condition, although it has been mentioned by certain writers who furnished case reports. In the authors' opinion it is questionable whether or not such jejunal ulcerations are indeed primary and not secondary to or coincident with unrecognized infection or ulceration in the stomach, duodenum or gall tract and all of them derivable from some focus of infection in the respiratory, nasal and paranasal tracts, or from foci in tonsils, gums or teeth.

Gastrojejunal or so-called marginal ulcers are seen much more frequently, and here the evidence of there being secondary infections to foci higher up becomes much more apparent. The usual etiological explanation of the development of these ulcers has been ascribed to the use of nonabsorbable suture material used in the performance of the gastro-enterostomy or as a result of trauma from improperly applied clamps. Conceding these possibilities, the authors nevertheless feel that some additional emphasis should be laid upon the infectious origin of marginal or gastrojejunal ulcers which so closely follow the primary operation, and they believe that in a majority of the cases the two factors mentioned above will represent retarding influences in the healing of the lesions without themselves being the producing cause of the ulcer. They, therefore, believe that the great majority of ulcers are infectious in origin, or, more properly speaking, are produced as a result of infection acting upon mucosal zones of diminished resistance.

One should constantly remember that before any ulcer case is definitely taken up for treatment, whether this treatment be medical or surgical, an earnest attempt should be made to determine the etiological type and source of the ulcer with which one has to deal. It would be very foolish to withold specific therapy and instead to operate on a syphilitic gastric or duodenal ulcer, ignoring the fact that the ulcer was being produced by lues. Again, and most importantly, it is never wise to operate for ulcer of infectious origin and perform a gastro-enterostomy, and expect to see the patient get well if we leave behind the primary infection in purulent tonsils or a "dirty" septic mouth. It is often in this manner that postoperative marginal ulcers are created.

The authors remember how forcibly this fact was brought home to them in a comparatively recent case which occurred in a prominent Philadelphia surgeon. About six years ago he was operated for the relief of a duodenal ulcer on the posterior wall of the duodenum which was causing partial obstruction. A gastro-enterostomy alone was done. He made a poor response to surgery, and within a few months symptoms of a slightly different type reappeared, kept on progressing and finally took him back to the operator who said there was nothing more to be done. He then went to a most important surgical clinic and they corroborated the first surgeon's opinion. He continued to suffer with ulcer symptoms, but during the war he was ordered to France and was forced to eat "Army chow" that was perfectly good for any healthy individual, but bad for a gastric ulcer case. He continued to get worse, and on his return went to the same clinic and was taken up for operation. He was found to have a marginal ulcer at the distal lip of the stoma. The operator closed the gastro-enterostomy, did an end-to-end anastomosis of the jejunum, went back to the duodenum, demobilized it and did a Finney pyloroplasty, and let him go. They, too, failed to hunt for, or at least failed to find, the primary focus which had produced both ulcers. Well and good! The patient made a stormy post-operative recovery and within a few months redeveloped the same type of postmeal hunger distress and pyrosis, but modified as to their time interval on account of the changed motility due to the rapid emptying of his stomach following the pyloroplasty. At this point it was easily demonstrable by tubing the patient that he had a hemolytic streptococcic infection in the duodenum and stomach. Where was it coming from? This meant careful search for focal factors of infection above the stomach. The teeth and gums were negative. His sinuses and bronchial tree gave no clues, and casual inspection of his pharynx and tonsils showed nothing visible on half a dozen examinations, except that the posterior pillars of the tonsil were bound down and the tonsil could not be pressed forward until the posterior pillars had been freed, when nearly a teaspoonful of pus welled out, foul and offensive, and grew out pure cultures of hemolytic streptococci. Here then was the presumable source of all his trouble. The tonsils were removed and a vaccine prepared. It took a year to get this man back to health, during which time he had much topical disinfective treatment to his stomach and duodenum, coupled with general management and the use of his vaccine. His hemoglobin at its lowest point was down in the thirties; now it is in the nineties. His weight was forty pounds less than his normal. Now he is back above his usual normal weight. His strength and efficiency in work were badly reduced. Now he is doing about 90 per cent. of his work and getting away with it.

In the same way a similar analogy could be brought forward to explain simultaneous gastritis, duodenitis with or without ulcer, and infective cholecystitis, appendicitis and sigmoiditis. The authors wish they could drive home the point more forcibly that the same infective agent can produce multiple lesions, coexistent in the gastro-intestinal tract, and they believe that all, both surgeons and internists, are equally culpable in neglecting the search for the primary and all secondary focal points of infection.

The site of gastrojejunal ulceration is generally along the margins of the stoma. The posterior margin is more commonly affected and the distal lip of the stoma is more frequently involved in cases of secondary causation in which the ulceration is being produced by infective agencies draining through the new gastric opening, whereas the proximal lip of the stoma is more commonly attacked in cases which show concomitant infection in the gall-bladder or bile-ducts. Less frequently the ulcer will be in the jejunal wall opposite to the stoma and overlying the attachment of its mesentery.

There has been very little work done in regard to the actual pathology of these ulcers. They are more apt, however, to be small, linear and more superficial than those usually occurring in the stomach and duodenum. But, nevertheless, they show very little tendency to heal. The authors' opinion in this respect is based upon their experience with clinically recognizable marginal ulcers which are most resistant to treatment. It is possible, however, that in this zone, just as in gastric and duodenal ulceration, the more superficial ones heal readily before they have given rise to interpretable symptoms or objective findings.

Specific Conditions.—Pain is of frequent occurrence, is constant as to its duration over many consecutive weeks and with less of the spontaneous remission common to duodenal ulceration; it is more inconstant, however, as to temporary relief through food-taking or the use of alkaline powders as in gastric or duodenal ulceration. The most severe pain occurs as in duodenal ulcer from one to three hours after eating and the time interval seems to bear a certain relation to the motor function of the gastro-enterostomy. The pain has been generally thought due to the corrosive action of a hyperacid gastric juice and at times assumes the suggestiveness of a pyrosis. In certain cases, however, the pain is not due to hyperacidity, but, on the contrary, is found to be caused by a hyperalkalosis. For in fractional gastric studies on some such patients it will be seen that at some point after the forty-five-minute period, bile is regurgitated back into the stomach in larger and larger quantities and the gastric acidity curve rapidly drops to the alkaline side. With this regurgitation will be found the direct diagnostic evidence of blood, pus, bacteria and inflammatory débris and the beginning of pain is coincidental with the regurgitation of bile and increases in severity with the increasing bile reflux. One would assume from this that the alkalinity is developed from the bile itself. This is probably not true, however, inasmuch as bile from the gall-bladder and gall-ducts freshly recovered from the duodenum, the authors have found to be acid in reaction, varying between twenty-five and forty-five degrees of acidity when titrated to phthalein. Therefore the alkalinity of bile found in the jejunum is probably due to its admixture with the succus entericus. That this phenomenon is not a physiological compensatory one designed to overcome a supposed hyperacidity is evident by the therapeutic test of administration of hydrochloric acid which will often serve to relieve the pain where alkalis aggravate it. The authors have seen certain patients who have got greatest symptomatic relief from this measure, and, in addition, they have found that lying flat on their back on the floor or a hard couch is the posteral position most apt to relieve their distress. This position may in such cases prevent the regurgitation of bile across the marginal ulcer and into the stomach.

The preoperative diagnosis can be most easily and accurately made by means of the duodenal tube and the microscope. X-ray as a rule will not disclose or differentiate this pathological lesion.

Very little has been accomplished in the treatment of postoperative marginal ulceration by any method of medical management, and surgical interference probably offers the greatest prospect of permanent correction, although the operative technic is most difficult and the mortality risk is high. In no case, however, will the infective type of this condition be likely to be permanently cured by either surgical or medical procedures if the primary source of the infection is unrecognized or disregarded as has been stated above.

Typhoid and dysenteric infections are taken up under their special headings in other sections of TICE'S PRACTICE OF MEDICINE.

Tuberculosis.—The primary type of ulceration is rare and is believed to result from infected milk. The secondary type is the more common of the two and this is the ulceration usually seen accompanying tuberculosis and results from infection from the swallowed sputum, or may be hematogenous in origin.[37] It is characterized by colicky pains, diarrhea and fever. Or there may be extensive ulceration without marked symptoms. Early involvement is frequently accompanied by constipation with abdominal discomfort and bloating although an outbreak of diarrhea may occur later. Usually pain soon follows and may be most marked several hours after meals when the bowel contents is beginning to reach the cecum causing colonic peristalsis to become more active. This may continue or alternate with constipation but usually there is a tendency to overactivity of intestinal motility and the diarrhea becomes progressively worse with increase of the colicky pains and distress. Hemorrhage rarely occurs to any great extent. Examination of the abdomen elicits tenderness usually in the right lower quadrant and the ascending and transverse colon may be distinctly palpable. Usually, though, where matting of the bowels has occurred from peritoneal adhesions, the abdomen will feel "doughy" to the palpating hand, even in the absence of peritoneal fluid. Masses simulating tumors and localized abscesses may be felt in the more advanced cases.

In an excellent paper by Brown and Sampson[8] the subject of intestinal tuberculosis is studied most carefully and they point out the difficulties of making a correct diagnosis except by means of the roentgen ray. They find that with few exceptions they are able to diagnose tuberculosis of the cecum by the absence of barium at all times in the cecum because of spasm with marked hypermotility. "The smooth haustral sacculations were absent and the bowel had a distinctly irregular and ragged appearance."

Besides the ileum and cecum, the stomach and jejunum, the colon and rectum may become involved. This type of ulceration, when seen in the jejunum or upper ileum, is usually round or ovoid and at right angles to the lumen of the bowel. It increases in size by extension along the lymphatics and tends to surround the bowel, involving a part or all of a Peyer's patch. It is found in all stages from the miliary tubercle to caseation and sloughing, involving the layers of the bowel down to the serosa, which usually shows nodular cords following the course of the lymphatics (tuberculous lymphangitis) and with tuberculous lymphadenitis. There is a tendency to matting of the bowels from plastic peritonitis which may be set up either from an extension of the process from within the lumen of the bowel or from a pelvic tuberculosis (tubo-ovarian), and as a consequence the ulcerative process rarely perforates. Where healing occurs stenosis is common.

Simple Ulcerative Colitis and Catarrhal Ulceration.—Bassler[16] has shown that a diffuse ulceration of the epithelial surface is possible and he has traced the causative agent to the Bacillus colon group. Under certain conditions an extremely virulent strain of Bacillus coli may be developed resembling the dysentery bacillus, and producing symptoms somewhat similar to it.

Stercoral and Decubital Ulcers.—In the authors' experience this type has been mostly confined to the elderly and frequently bed-ridden patients. Through lack of the proper motion of the body the coils of the intestine are not changed in their position sufficiently to aid the weakened bowel musculature to pass on its contents completely enough and stagnation occurs. Hardened masses of feces form in the colon and become agglutinated to the mucosa and when they are eventually pushed along through purging, or what not, ulceration quickly follows through secondary infection attacking the denuded bowel wall. Diarrhea and pain may be extreme then and there may be severe hemorrhage. This or perforation may close the chapter for the patient after years of suffering.

Space will here be taken to briefly discuss the etiology, symptoms and pathology of some of the rarer forms of ulcerations.

Traumatic Ulcerations—Burns.—These are frequently rapidly fatal from profuse hemorrhage. They give pain and watery diarrhea as the chief symptoms. Their duration is from a few days to two or three weeks. These ulcers may be either single or multiple and usually involve the duodenum, rarely the stomach, and probably result from toxic substances in the bile.[16] They show little tendency to heal and are usually sharply defined and extend down to the deeper coats of the bowel.

Spinal injury and multiple neuritis will sometimes be accompanied by intestinal ulceration. This type of ulcer is very similar to that seen in burns and is thought to be due to injury of the nerves which results in blocking the trophic impulses to the intestinal mucosa, perhaps producing an endarteritis with necrosis.

Thrombotic and Embolic Ulcerations.—Here the pain is not a marked feature nor is the bleeding profuse, but watery diarrhea is the important symptom. Any part of the gastro-intestinal tract may be involved by this type of ulceration, but it is seen more usually in the jejunum, ileum and colon. It is nearly always associated with abscess formation somewhere in the body, malignant endocarditis or pyemia. Together with the ulceration there is usually embolism in other parts of the body with infarction of the lung, spleen, liver, brain or kidney.

Amyloid Ulceration.—The symptoms are those of the embolic type just described but there is usually here a characteristic history which points to diagnosis. These ulcers may be seen anywhere in the bowel, are usually multiple and are rather rare. Associated with ulceration, there is amyloid degeneration of the mucosa contiguous to the ulcer. They show no tendency to heal. They are always associated with amyloid degeneration of the liver or spleen and result from long-continued suppurative processes, tuberculosis (usually of the bones), blood dyscrasias, and malignancy.

Anthrax.—This condition results from eating infected meat or as a secondary infection from a malignant pustule. Constitutional symptoms together with profuse diarrhea are marked. This is a rare type. With the lesions as described under acute enteritis there may be areas of necrosis with surrounding edema of the mucous membrane, in which the anthrax bacillus is found.[13] An associated hyperplasia of the spleen and lymph-nodes is usually found.

OTHER TYPES.—The *syphilitic* ulcer is uncommon, except in the rectum, but has been described as occurring in the small bowel. It is characterized by its markedly indurated edge surrounding a smooth grayish area from which the epithelium, which previously overlay a gumma, has been lost. There is a marked tendency to stenosis which occurs as healing progresses. The ulcers of *follicular* enteritis and colitis are described under acute enterocolitis, and are small defined ulcers, often multiple, in either the small or large bowel. There are also *ulcers in acute infections* (botulism, sprue, erysipelas, varioloid and diphtheria); in *chronic infections* (pellagra and leprosy); and in *constitutional disorders* (purpura, cachexia of cancer, leukemia, nephritis, scurvy, and gout). *Toxic ulcers* are the result of poisoning. There are also those ulcers seen infrequently in gonorrheal infection and those resulting from breeding of larvæ of flies (myiasis).

Diagnosis.—The important thing in arriving at a diagnosis of this condition is to determine the causative agents. The history must be investigated and the possibilities of a specific infection considered. As pointed out under the considerations of symptoms every method of investigation known to the gastro-enterologist should be utilized in isolating a specific infecting organism if present, and, of course, the most important of these are the protozoal parasites and the organisms which cause dysentery. Unless one can rule out these specific infecting organisms, it will be useless to endeavor to treat the patient, for therapeusis must be directed against them if found. Next to the type of infection, the diagnosis of the location of the lesion becomes important when one comes to consider treatment.

Among the acute diseases which one has to rule out, typhoid is perhaps the most important. Here the temperature, splenic enlargement, rose spots and Widal reaction, together with blood and stool-cultures, should make the diagnosis.

Neoplasms and other tumors must be considered where one can palpate a mass within the abdomen. While such tumors may themselves set up a chronic colitis, they do not frequently lead to actual necrosis of the bowel wall.

Localized abscesses, chronic appendicitis, adhesions and gall-bladder disease must also be considered, but will hardly be confusing if one is careful in his observations of the stools and in other physical and technical examinations.

Pernicious anemia is mentioned here so that it may be kept in mind. It is hardly likely that one would go far astray if he is familiar with the blood-picture in this disease, as it is so fairly characteristic that it is not likely that any ulcerative process would simulate the blood-picture.

The tuberculous type of ulceration is practically always accompanied by pulmonary tuberculosis which has antedated the gastro-intestinal symptoms by many months. Again, as pointed out in symptomatology, this is a difficult diagnosis to make and unless the x-ray is relied upon the early ulcerative processes may be readily overlooked. The toxemia of tuberculosis alone will very frequently give gastro-intestinal symptoms closely simulating those of ulceration. In these cases the roentgen ray seems to be holding out a hopeful field in diagnosis.

Treatment.—General Measures.—In no other gastro-intestinal condition does **rest** become such an important factor in recovery as in this one. There is no form of ambulatory treatment that the authors know of that they can recommend. This is true both of the acute and chronic types of ulceration. This rest should be complete and nothing short of **total confinement in bed** will answer if one is to get results from treatment. The **food** (see Chronic Enterocolitis) in all cases should be **bland and nonirritating**. This usually means an **almost complete carbohydrate diet** except in the case of sprue, where a lean meat diet seems to be the only one that will produce results. It frequently means a **semiliquid starch diet**, or, where vegetables are allowed, the cellulose element together with hulls and shells should be removed by straining through a coarse cloth or a colander. **Milk, predigested with caroid or pepsin, or boiled or carbonated or sour**, may be used where it does not cause discomfort. If there is no tendency to fatty diarrhea, **cream, olive oil**, and **butter** are valuable adjuncts in keeping up the nourishment of these patients while on this restricted diet.

Fresh air and sunlight are at all times valuable in the treating of disease but especially in tuberculous ulcerations they become notoriously important elements in the treatment. Locally, **sunlight** applied to the **abdomen** over long periods of time and preferably in high altitudes, where less of the actinic rays have been filtered out by the atmosphere, has been known to be extremely beneficial.

Vaccines, especially those of the colon groups, have been used to great advantage, as well as those prepared from other infecting organisms.

With **intubation**, by means of the various intestinal tubes that have been referred to previously, various chemical substances can be introduced. A 0.5 per cent. **calcium carbonate solution** in 1000 c.c. (33.81 fluidounces) quantities has been used as daily instillations by Einhorn [29] with good results in a case of chronic ulcerative colitis, presumably of the catarrhal type, as no specific organisms were mentioned in his report. Likewise Palefski [28] and Jutte [27] have been using for this purpose **solutions of sodium chlorid, sodium sulphate, magnesium sulphate, soda bicarbonate** and **plain water. Insufflation with oxygen** has been used, particularly in infections known to be due to the anaërobic types of bacteria. It may be possible by the use of these tubes to use **mercurochrome** for its local transient action, as by this tube method, one is able to flush out the bowel immediately after this antiseptic has been used, and thus prevent its absorption into the system. There is still much to learn in the use of this new chemical. According to Davison,[38] who has been doing experimental work on dogs with mercurochrome applied to the gastro-intestinal tract, it is non-toxic in moderate amounts (30 c.c. [1 fluidounce] of a 1 per cent. solution) except for the production of purging similar to that caused by calomel. **Sigmoidoscopy** allows one to make direct applications to ulcerated areas by application of such solutions as **copper sulphate,** 30 per cent. in water, **silver nitrate,** 10 and 20 per cent., and **insufflations of calomel powder** and **tannin. Irrigations of the bowel** are possible, but perhaps not very useful. Those that have been used with some reported success are **thymol,** 1-2000 or 1-5000; **quinin,** 1-3000 or 1-5000; **silver nitrate,** 1-5000 or 1-10,000; and **kerosene.** Any of these may be injected in 500 c.c. (16.90 fluidounces) quantities in an endeavor to reach all parts of the colon. **Surgery** is rapidly coming to the fore in the treatment of tuberculosis and other types of ulceration and one should refer to the works on this subject for its definite indications.

HEMORRHAGE.—Intestinal hemorrhage from ulceration may require very active treatment. Here not only must the individual be physically resting, but the bowel itself must be put in splints, so to speak, by the use of **morphin subcutaneously. Hemostatic preparations and sera** should be used, hyperdermically or intravenously. **Ice** should be applied **locally** by means of the **coil** or **bag.** Surgery has no place in acute hemorrhage, but after the profuse hemorrhage has been stopped and, if continuous bleeding follow, then surgery may be needed to prevent further bleeding. It may first be necessary to give intravenously from 100 to 250 mils of 25 per cent. glucose in sterile normal saline solution. Or a blood transfusion may be required providing it be possible to type the blood and procure a donor whose blood is of the same type. There are few medicines that can be taken by mouth that will have any effect on bleeding. Ergot is mentioned only to be condemned. **Lead acetate** has been found useful at times, as has **tannin.**

STENOSIS.—Stenosis and adhesions causing obstruction can be dealt with only by **surgery.**

PAIN.—The best means of controlling pain is by means of a **hot compress** as recommended by **Priessnitz.** This consists of several thicknesses of flannel wrung out in hot water applied to the abdomen and covered with oiled silk and overlaid with a hot water bag or electric heating pad. Where heat does not give relief, **an ice bag** may be tried.

DIARRHEA.—This is a difficult symptom to control in well-advanced ulceration. **Opium, chalk, kino** and **bismuth** may be tried, but are frequently found wanting.

Special types of ulceration require special measures. The dysenteric types are taken up in another chapter, so will not be considered here. No discussion will be made of typhoid or the rarer forms of ulceration that are seen in leprosy, botulism, etc. The authors would like especially to mention here the use of **iodoform, iodin, and creosote** in the treatment of tuberculous ulcerations. It seems that these preparations, if they can be topically applied to the region in which the ulceration is occurring by means of the intestinal tube, may possess some merit in furthering a cure. Fishberg[89] has reported the use of 5 per cent. solutions of **calcium chlorid**, of which 5 c.c. (1.35 fluiddrams) is injected intravenously, as being very efficacious in controlling the diarrhea and pain in early cases. Stress should be laid on the use of **sunlight locally applied** to the **abdominal wall** as being an important measure in the cure of these early cases. Syphilitic ulcerations, strange to say, are not readily amenable to **mercury** and **neosalvarsan.** However, these drugs must be routinely given and the dosages forced if one hopes to obtain any results at all. The indications in the embolic, amyloid, decubital, and other types of ulcerations are essentially those as outlined under general measures.

Prognosis.—Unfortunately the prognosis is usually good or bad, depending upon the type of infecting organism. With the dysenteries and proper medication the prognosis is fairly good. The amyloid, embolic, follicular and traumatic ulcerations usually prove fatal, as do many of the other types. In tuberculosis there is more and more hope that surgery[86] will eventually be the remedial agent of great value. It is still in its infancy and as time goes on the authors believe that the surgeon will be able to extend a more hopeful prognosis to these poor sufferers. Perhaps the newer methods which are rapidly coming to the fore, especially those designed along the lines of topical application to the lesion by means of the intestinal tube and sigmoidoscope, may give better results in the treatment of ulcerative processes than have heretofore been possible. As the result of healed lesions in which stricture and adhesions have formed, surgery becomes the only resort.

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CHAPTER XXI

APPENDICITIS

By John A. Lichty, M.D.

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Definition.—The term appendicitis includes the inflammatory conditions which have their origin in the structures of the *processus vermiformis* and which may extend to its peritoneal covering and also to the surrounding tissues and organs. This inflammatory condition may be immediately *acute,* or it may be of slow process producing only occasional slight disturbances and resulting in a more or less *chronic* lesion.

Etiology.—The factors which enter into the cause of appendicitis are numerous and varied, and, depending upon whether the one or the other factor predominates, the character of the disease varies greatly. These factors may be classified under *predisposing, essential* or *infectious,* and *contributing* causes.

Predisposing Causes.—The embryological development of the appendix and the anatomical structure and relations thereof are not the least of these factors. During embryonical life all the structures which are given to the gut are also given to the appendix, which begins to be separated about the eighth week. About the time of birth the intestinal contents begin to enter from the ileum and the cecum takes upon itself sacculation, but the appendix retains all the longitudinal muscle intact without separation. In the cecum they separate into the three bundles which characterize the large bowel. In the same way, all of the structures, including glandular and nervous, which in uterine life go to make up the intestinal canal, are segregated to the vermiform appendix.

In the bowel, however, they carry with them a necessary physiological function; in the appendix, there is no such physiological activity thus far recognized. An exhaustive discussion of this subject may be found in the admirable work of Kelly and Hurdon.[1] It would seem natural to conclude, therefore, that an organ which is equipped with all the structures and elements necessary to normal function and yet apparently does not functionate, would be likely to harbor disease processes.* The anatomical relations of the organ also predispose it to disease.

Relation to the Cecum Anatomically and Physiologically.—This relation is through the structures which are common to both organs, but the lumen which forms the communication between the two organs may vary considerably and yet be within the normal. There may be such a disposition of the mucous membrane at the point of communication as to serve the purpose of a valve (Gerlach's valve). There may also be a certain physiological spasticity of the circular fibers of the cecum at the point of communication with the appendix as to form a temporary closure, locking up contents from the bowel for a time. These may be of such a degree as to produce or favor disease. This irregularity of the sphincter action has been noted particularly in x-ray examinations of the gastro-intestinal tract. It has diagnostic value.[3]

On account of the rudimentary structure of the appendix and on account of the want of any definite function, the appendix about middle life, or soon thereafter, undergoes an involutional change, so that finally it presents the appearance of a hard fibrous cord with scarcely any lumen. This has been called *obliterating appendicitis.* It has been suggested that this change may occur also as the result of an inflammatory reaction. The process usually begins at the tip and extends toward the base. Occasionally it is found localized some distance back of the tip, while the tip is still apparently normal. This may be the result of a localized slow inflammatory process which may eventually lead to an acute exacerbation, producing the symptoms and signs of acute appendicitis.

Relations to Other Organs.—The appendix usually has a mesentery, and at times this may be long enough to allow of a wide range of movement, especially of the distal end. When there is no mesentery, or when it is short, the appendix may be in close relation to the head of the cecum lying anteriorly, posteriorly, or to the outer or inner sides. Any of these positions may be the determining cause of disease, but when the mesentery is long and the tip freely movable it may engage in an inflammatory process which is primary either in the pelvis on either side, in the gall-bladder region, in the region of the stomach or pylorus, or in the opposite side of the abdomen. In some of these positions the appendix may become kinked in such a way as to favor disease.

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* In 1912 Sir Arthur Keith[2], Conservator of the Museum of the Royal College of Surgeons, London, in a magazine article, said, "For many years, the appendix vermiformis has been regarded as one of the vestigial structures of man's body, an opinion which has prejudiced us against any real endeavor to discover its nature and function. Twelve years ago, Prof. R. J. A. Berry, on the evidence he then produced, came to the conclusion that the appendix should be regarded as a specialized part, not a retrograde part, of the cecum, and that is the conclusion which every one must reach who makes an impartial survey of our knowledge of the comparative anatomy, and of the evolution of the human ileocecal region."

AGE.—Appendicitis is a disease of early adult life. If the facts were known it is quite likely that it could be said that in the acute or initial attack it is largely a disease of children and that of very young children. The diagnosis of the lesion in children is fraught with considerable difficulty on account of the want of intelligent coöperation on the part of the patient. Pediatrists continually report the difficulties with which they must contend, and speak of the slender evidence upon which they are occasionally compelled to make their diagnoses. McCosh [4] reviewed 1000 cases of appendicitis in children and found only 4 cases under the age of two years. E. G. Alexander [5] reports 500 cases in children operated in John B. Deaver's clinic at the Mary J. Drexel Home, Philadelphia. Children of all ages are admitted into this Home, but very few enter after the age of twelve. Seventy-nine were twelve years of age, 71 were ten years of age, 48 were eight years of age and so on down to 4 who were only two years old. The *seasonal* incidence in these cases seemed to favor May, June, July and August, though February had a high incidence. While seasonal influence may be only a small factor in the incidence among adults, among children who are afflicted with "summer complaint" a certain part of the year, or "colds" another part of the year, the increase in July, August and February may be of some significance. The gradual increase of cases in children from 4 at two years to 32 at six years, and 79 at twelve years, is strikingly in agreement with the increasing incidence in adults up to twenty-five years of age.

In the writer's own acute cases which were operated, the ages were as follows:

Between 0 and 9 years	4	(youngest 8 years)
" 10 " 19 "	11	
" 20 " 29 "	19	
" 30 " 39 "	20	
" 40 " 49 "	21	
" 50 " 59 "	7	
Over 60 years	1	(aged 62 years)

SEX.—It is generally considered that the male sex is more likely to suffer from appendiceal disease than the female. The statistics available on this point are not altogether convincing. They come largely from the surgical clinics. The writer's opinion based upon his own cases, acute and chronic, numbering 1430, of which 500 were operated, is that the male is more likely to come promptly to an operation, probably with the first acute attack; whereas the female, because of the menstrual epoch, or on account of existing pregnancy, or from the fact that she bears pain comparatively more easily than the male, is more likely to defer operation. Operation for the acute attack is therefore comparatively more frequent in the male and for the *chronic condition,* in the female. This is borne out by the following statistics:

Symptomatology.—The symptoms of acute appendicitis are usually quite definite; those of chronic appendicitis are frequently indefinite. The two conditions will therefore be discussed separately.

ACUTE APPENDICITIS.—*Symptomatology.*—Usually the first and chief symptom is *pain* in the epigastrium. This may be of a cramp-like nature, and, unless peritonitis or perforation take place, is rarely of sufficient severity to demand a hypodermic. Later it becomes more severe and finally it is localized in the right lower quadrant at McBurney's point. This localization may be quite prompt, or on the other hand may not occur until the end of 36 or 48 hours. In fact, unless the patient is seen by a physician it may not be known to have localized in the right lower quadrant and may be considered as having been only an attack of "acute indigestion." The pain may be referred to the pelvis, or to the opposite side of the abdomen, or up into the right groin. When it localizes in the right lower quadrant, the pain in the epigastrium disappears. *Nausea* and *vomiting* appear sooner or later, depending upon the character of the meal taken previously or upon the severity of the attack. Because the vomitus contains undigested food, the previous meal is often thought to be the cause of the attack. Of 83 acute cases operated 72 had nausea during the attack and 56 vomited. The bowels were constipated in the large majority of cases. Only 7 of 83 cases operated had diarrhea. *Pulse* and *temperature* at first may be undisturbed, but as the attack progresses the pulse rate gradually increases and there may be a slight elevation of temperature. Depending upon the course which the case pursues, the temperature may (1) subside in 48 or 72 hours as in a mild form, or (2) increase gradually and become of a hectic type as local peritonitis and abscess formation take place, or (3) the pulse may become very rapid and the temperature fall suddenly with a rise as perforation and general peritonitis occur. In 59 acute cases the temperature was under 100° F. (37.77° C.); in 19 it was between 100° F. (37.77° C.) and 102° F. (38.88° C.); and in 5 it was over 102° F. A chilly sensation or an actual chill may occur at the beginning or during the attack. This is of considerable significance. The *leukocyte* count in mild cases, and early in the severe ones, is normal or only slightly increased. Later there is a gradual increase. If leukocyte counts are made early and frequently repeated, for instance every 2, 4 or 6 hours, the information gained will be of considerable value in determining the course and severity of the attack. The increase is in the polymorphonuclear element largely. A leukopenia early in an attack may indicate a virulent infection; and later in the attack, especially when the temperature and pulse remain up, may mean that the condition is particularly grave. The information gained from one or two leukocyte counts only during an acute attack is of comparatively little value. Of 59 acute cases operated, 9 had leukocyte counts of 10,000 or less, 19 had from 10,000 to 15,000, 19 between 15,000 and 20,000, and 12 had 20,000 or over. It would seem that the average leukocyte count is somewhere about 15,000. Such information is of little value, however, as it is the gradual increase in leukocytes in a certain time which gives confirmatory evidence. The patient with an acute attack, and before the pain localizes in the right lower quadrant, may be very restless and insist upon moving about. As soon as the pain localizes in the right lower abdomen, he prefers to lie on his back, with the right knee flexed. Deep inspiration and coughing will aggravate the pain. The lower end of the right rectus becomes rigid as the inflammatory process develops. This may be noticed before the pain localizes in the right lower abdomen and may at first be rather diffuse; later, especially if the inflammation does not spread to the peritoneal covering, the rigidity becomes definitely localized. On the other hand, if a general peritonitis supervenes, the rigidity accordingly spreads to the whole abdominal wall.

Course and Termination.—If there is a mild "catarrhal" inflammation the symptoms will subside in three or four days without further disturbance until the next attack. If the inflammation spreads to the *peritoneal surface,* but without pus formation, the attack may be concluded in eight or ten days and no symptoms follow excepting those of rather indefinite digestive disturbances from time to time, or soreness in the right lower abdomen upon physical exertion or over-exercising. Because of possible adhesions, subsequent attacks are more likely to occur. If ulceration and necrosis take place, *local abscess formation* may occur. Under such circumstances, tumefaction takes place in the right iliac fossa. This may lie immediately under McBurney's point, or extend down into the pelvis, or upward, back of, or to the right of the cecum. Occasionally at operation, a perforated appendix may be found in the abscess cavity. This occurred in 4 of 83 cases operated. The abscess sac may rupture and produce a general peritonitis. And finally, on account of rapid necrosis, a diseased appendix may suddenly *rupture* and produce a *general peritonitis.* This is usually the cause of death in patients not operated.

Complications.—These come on as the result of the extension of a suppurative process or as the result of absorption direct from the focus of infection, producing pyemia. In the event of one or the other there may be a *pylephlebitis, a subphrenic abscess,* or *pulmonary embolism.* Neuhof [10] reports 15 cases of nonsuppurative subphrenic peritonitis complicating appendicitis. These were collected from the Second Surgical Service of Mt. Sinai Hospital, for the years 1904 to 1910, which included 972 cases of acute appendicitis. All recovered. Hematuria is a confusing complication which fortunately does not occur frequently. Nore, Josser and Fayol [11] report 3 cases of their own and have collected 28 more from the literature. According to these authors it may be due to direct extension from appendix to right ureter or "through reflex influence from appendix to kidney." Guy L. Hunner [12] calls attention to the fact that acute attacks of appendicitis may begin with referred pains in the bladder region, and accompanying symptoms of cystitis, even accompanied with pus and blood in the urine. *Thrombosis* of the femoral veins, especially the right, is an occasional complication, but this is more likely to occur after operation for appendicitis.

Diagnosis.—The diagnosis of acute appendicitis depends largely upon the history of the case and the physical examination. Any pain in the abdomen, especially if it begins in the epigastrium and is later referred to the right lower quadrant, where tenderness is found directly over McBurney's point, and associated with nausea, must be considered as that of an acute appendicitis until the diagnosis is confirmed or until it is proven to be a pain from some other cause. This is a safe position to take in all these cases because appendicitis is the most common inflammatory condition in the abdomen in persons below middle life. According to W. McAdam Eccles[18] the most frequent abdominal conditions are as follows: (1) Acute appendicitis 70.7 per cent.; (2) intestinal obstruction 18.1 per cent.; (3) perforations 7.2 per cent.; (4) cholecystitis 2.4 per cent.; (5) torsion of pedicle 1.1 per cent.; and (6) acute pancreatitis 0.5 per cent.

Temperature, pulse and leukocyte observations should be made frequently but they should not stand in the way of, or delay plans for, consultation with a surgeon or for preparation for an operation.

Certain acute conditions in the abdomen should be kept in mind in making a diagnosis. Of those rarely associated with a temperature are:

(1) *Dietl's crises,* which may result from the kinking of a ureter in a movable kidney.

(2) *Stone in the right kidney or ureter.* This should be excluded by the absence of pain referred to the genitalia and of red blood corpuscles in the urine. An x-ray examination may reveal the stone.

(3) *The abdominal crises of locomotor ataxia.*

(4) *Acute obstruction of the bowel.*

(5) *Gall-stones.*

Except for acute obstruction of the bowel these conditions should be easily excluded. Of those acute conditions associated with a temperature there should be excluded:

(1) *Inflammatory conditions* in the pelvis especially those of the right tube and ovary. A careful pelvic examination should be sufficient to differentiate.

(2) *Typhoid fever* may be associated with pains in the abdomen similar to those of acute appendicitis. The slow pulse, the constant leukopenia, and the presence of rose spots and of an enlarged spleen should be noticed. However, it must not be forgotten that occasionally an acute appendicitis may occur in the course of typhoid fever.

(3) *Acute hemorrhagic pancreatitis* should be considered, especially if the pain is severe. But this is an exceedingly rare condition and is associated with definite tenderness in the upper abdomen. It has all the evidence of an acute abdomen requiring immediate surgical interference.

(4) *Meckel's diverticulum.* Unless there is some outstanding factor or peculiarity in the symptom complex, or a congenital defect in the patient, or a history of anomalies prevailing in the family, an acute inflammation of a Meckel's diverticulum cannot be differentiated from acute appendicitis. Fortunately this is of little significance, as both conditions call for the same treatment—surgery.

Occasionally a lesion above the diaphragm such as *lobar pneumonia*, or *diaphragmatic pleurisy* will be accompanied with pain in the lower abdomen and unfortunately now and then such patients have come to operation for appendicitis. This only emphasizes the fact that a diagnosis of acute appendicitis should not be made before a thorough and complete physical examination has been made. This should, of course, include an examination of the blood and urine. Another condition which occasionally simulates acute appendicitis is the abdominal form of *Henoch's purpura*. Attention was called to this in Osler's[14] article on angioneurotic edema. Dr. H. H. Donaldson[15] and the writer reported 2 such cases which were admitted to the Mercy Hospital, Pittsburgh, to Dr. J. J. Buchanan's service for operation for appendicitis. The patients had abdominal colic, vomiting and bloody stools and also pain in the joints. Large purpuric spots were noticed here and there. The patients were transferred to the medical department where the symptoms gradually subsided.

Chronic Appendicitis.—It is an open question as to whether chronic appendicitis can occur without a previous acute attack. From the nature of things it must be largely academic, and may be answered one way by the clinician and another by the pathologist. According to Aschoff and others every case is the result of a previous more or less acute attack. This is largely the view of the pathologist, who does not think of the condition so much in terms of symptoms as of a healed inflammatory lesion. Whether this can occur without any immediate symptoms is a question which could be discussed interminably but to very little profit. Certain it is that the patient's statements, however faithfully given, are not always reliable, for he is inclined to forget the exact symptoms which he experienced years ago when the lesion may have been in its acute form. Time and again the writer has questioned patients as to previous attacks of "acute indigestion" and has given every opportunity and incentive for them to recall such symptoms as would point to the appendix, but to no avail. After the operation, when a lesion had been found at the appendix, probably half of these same patients could recall more or less clearly that they *did* have "some sort of an abdominal attack" years ago, while the other half continued to insist that the only symptoms they ever had were such as they gave, referring probably to one or other of the abdominal organs. This may be because attacks in infancy and early childhood are often difficult to diagnose, and the diagnosis, therefore, never was made, or the patient has really forgotten, or the physician does not exercise sufficient tact or did not have sufficient patience and persistence to develop the history.

It may be possible that a lesion may occur and run through an acute and a subacute stage to a chronic form without producing any noticeable symptoms, or at least symptoms proportionate in severity and magnitude to those of the acute type. But among clinicians this can be looked upon only as an opinïon of which definite proof has thus far been lacking.

Symptomatology.—The symptoms arising from and usually attributed to chronic appendicitis are naturally divided into two groups: (1)

Those referred directly to the abdomen, especially to the right lower quadrant, and (2) those referred to organs and systems elsewhere.

Of the first, the symptoms within the abdomen, the most constant is *pain* and *soreness* in the region of the appendix. This pain is aggravated by exercise, especially by walking and running. It may be so severe as to incapacitate the patient for a few days, and yet there are no other evidences of an acute attack. The soreness can always be elicited by the examining hand and unfortunately the patient frequently himself learns to develop pain by making pressure. It is always the cause of considerable anxiety. It may extend down into the right thigh. On the other hand, the symptoms may be entirely gastric with only slight soreness in the region of McBurney's point. Under these circumstances there may be only the symptoms of a "chronic dyspepsia" such as the discomfort of "gas" in the stomach, loss of appetite, coated tongue, constipation and occasionally nausea. Of 400 chronic cases operated 279 were constipated, 18 had diarrhea and 103 had regular, normal bowel movements; 230 had nausea at times and 115 of these vomited. Or again the symptoms may be definitely those of peptic ulcer with acid eructations, gnawing sensations in the epigastrium, and "hunger pains" relieved by taking food. Sir Berkeley Moynihan,[16] in his Address on Surgery before the British Medical Association, 1913, said, "There is now no longer any doubt in my own mind that the commonest site of a *gastric ulcer* is in the right iliac fossa. That is to say, in the majority of cases, where the symptoms would justify or compel a diagnosis of ulcer, the patient is suffering from a lesion elsewhere, and more often than not, from a lesion in the appendix."

Occasionally the bowels are loose and contain considerable mucus. John B. Deaver in his early teaching called attention to the significance of mucus in the stool in relation to chronic disease of the appendix.

Of the second, the *symptoms outside the abdomen*, much could be written but little can be said with any degree of certainty. The patient gives a history of one or more of the abdominal symptoms already described and adds thereto those of nervousness, weakness, headache, backache, hypochondriasis, and many others rather bizarre. It is frequently difficult and in fact impossible to recover from the tangle of symptoms the history of an acute attack.

Diagnosis.—This can be made with a certain degree of assurance only, depending upon whether a history of an acute attack can be developed. Without such a history, physical signs and laboratory tests must be taken at a considerable discount. Of 400 cases operated only 217 gave a satisfactory history of having had acute attacks of abdominal pain which could be interpreted as being of the appendix.

It will be found best, therefore, to make the diagnosis by exclusion. In this way it may be possible to eliminate certain diseases such as chronic peptic ulcer, "latent gall-stone," cholecystitis, stone in the right ureter or in the kidney, certain chronic pelvic diseases, the various ptoses of the abdominal organs, and certain so-called endocrine disturbances. To do this a thorough routine physical examination should be undertaken, which should be followed by test-meals, examinations of the stools, of the blood, including Wassermann test, and of the urine including kidney efficiency tests. An x-ray examination of the entire intestinal tract may be of great value, not so much in giving direct information of the appendix as in determining the presence or absence of disease elsewhere.

The physical examination should include a careful examination of the abdomen. Of the greatest significance is persistent tenderness at McBurney's point. This may be definite at one time and indefinite at another. It is best elicited by making firm pressure over McBurney's point while the patient is lying on his back with the shoulders elevated and the knees partly flexed. An indefinite tenderness may be developed into a more definite sign by making pressure with the index finger over McBurney's point and then asking the patient when he is in the above position to extend the right knee, hold the leg rigid and elevate it from the bed or examining table. The diseased appendix thus pinched between the approximating anterior abdominal wall and the rigid belly of the iliopsoas muscle, gives evidence of extreme tenderness. This sign was first suggested by Meltzer[17] and goes by his name. It is a procedure which is, of course, applicable only in subacute and chronic conditions. By inflating the large bowel with air (Bastedo's sign), or by exerting pressure downward over the cecum towards Poupart's ligament (Rovsing's sign), pain and tenderness may be produced in the right iliac fossa. Among the writer's cases of chronic appendicitis operated, 286, or 71.5 per cent., gave a definite Meltzer's sign. It is interesting to note that in the 400 operated cases only 23 had rigidity of the right rectus muscle.

By the examination of the abdomen it should be determined whether there is a partial or a general visceroptosis, as this condition frequently is accompanied with symptoms suggestive of chronic appendicitis, although the appendix may be normal. Disease of the genito-urinary organs, especially of the right ovary and tube and of the right kidney and ureter should be excluded before making a diagnosis. In an obscure case catheterization and x-ray examination of the right ureter may be justifiable. In the male patient the investigation is not complete without a rectal examination.

Occasionally the pain from a right sacro-iliac strain may be referred forward and produce pain and tenderness like that of chronic appendicitis. The aid of a competent orthopedist is therefore often valuable in suspected cases.

And finally, a routine examination of the teeth, tonsils, sinuses and eyes, together with an examination of the chest, should be followed out.

Laboratory examinations may not furnish any direct information in establishing the diagnosis of chronic appendicitis but they are of great assistance in establishing the diagnosis of other conditions which might suggest chronic appendicitis. The constant presence of a *gastric hyperacidity* in the absence of peptic ulcer is of considerable significance as it may indicate pathology elsewhere in the abdomen, and the most frequent location of that pathology is in the right lower quadrant. Of 263 chronic cases of appendicitis operated in which gastric analyses were made, 189, or 71.9 per cent., had a hyperchlorhydria; 47, or 17.8 per cent., had a hypoacidity, and 27, or 10.3 per cent, had an achylia. The cases with achylia were, in the majority, such as had a history of great chronicity and were much below their normal health and revealed at operation extensive pathology by way of adhesions about the appendix and cecum.

The effect upon gastric function of extragastric pathology, occurring in the abdominal cavity, has been commented upon by others during the past ten or fifteen years. Among these there are Graham and Guthrie,[18] Paterson,[19] Fenwick,[20] Moynihan,[21] and Lichty.[22]

A thorough examination of the stool should be made for parasites and for blood, and the presence of the latter must be interpreted. Occasionally intestinal parasites are responsible for the symptoms. In one of the writer's cases pin-worms were found in the appendix. In a number of suspected cases of appendicitis, successful treatment for intestinal parasites cured the patient. Finally, x-ray examination of the stomach and bowels should be made. Pfahler[23] says that the chronic appendix gives much roentgen evidence of value in diagnosis on account of: (1) Localized tenderness; (2) visualization of appendix; (3) fixation; (4) position; (5) kinking or angulation; (6) constriction; (7) abnormal retention; and (8) incompetent ileocecal valve. To this he adds later the visualization of *fecoliths*. To the author the demonstration of fixation (adhesions), angulation, constriction, and an abnormal retention of bismuth have been of considerable diagnostic value.

If, after the foregoing procedures, the diagnosis is still in doubt, a tentative course of treatment, hygienic, dietetic, and medical, based largely upon the symptoms may be undertaken for a period of probably two to four weeks. When this is done with the patient under close observation, especially if it is a patient who will coöperate, a satisfactory diagnosis may often result where otherwise it could not be made.

Treatment.—Strictly speaking, appendicitis, acute or chronic, is a surgical disease and should be treated as such.

PROPHYLAXIS.—From the etiology it is already recognized that "preventive" treatment, to be of any value, must necessarily begin in early childhood and must be continued throughout life. It should be directed largely towards the selection of proper food and the prevention and cure of constipation. In children, in fact babes, the necessity for regularity of daily bowel movements should be plainly emphasized. Cathartics must be avoided. Heads of households should provide such food for the table as is wholesome and from which it will be possible to select a **well-balanced diet.** The food should be properly prepared, and sufficient time must be taken to eat it. Regularity of meals is of prime importance. The present way of living is directly the opposite to almost every detail mentioned, and a heavy burden lies upon the family physician, the trained dietitian, and those social organizations which come in closest contact with the conditions as they exist at present. To what extent the incidence of appendicitis and its consequent chronic invalidism could be avoided by proper prophylactic measures no one can estimate.

ACUTE APPENDICEAL ATTACK.—Immediate operation is the safest procedure. If operated in the early stage of the disease the death rate, according to the statistics of many clinics, is below 1 per cent. The mortality increases in direct proportion to the delay of the time for operation. There can be only two reasons for not operating as soon as a competent surgeon is called to the case: (1) Uncertainty as to diagnosis, and (2) advanced stage of the attack, when conditions of the peritoneum, local or general, are such that waiting for a definite "walling off" of the lesion or for an "interval operation" might be safer. In the first instance, the physician and surgeon need only agree as to whether there is an "acute abdomen"; in the second, it requires the most careful judgment of both.

MANAGEMENT BEFORE OPERATION.—What should the attending physician do until the surgeon arrives? This question is answered best by indicating what he should *not* do. **No cathartics**, however mild or severe the symptoms! It is safe to say that castor oil and like cathartics have increased the mortality of acute appendicitis more than any one other agency. **No enemas!** They only provoke the symptoms and may be dangerous. **No food!** The patient in almost every case will have just had a sufficient meal and can safely "starve" until the diagnosis is made, or until the acute symptoms have subsided. **No opiates**, until the diagnosis is made! It is an exceptional case in which opiates are indicated at any time. **Local applications**, such as ice-bags and counterirritants, should be withheld until the surgeon has made his examination. If nausea and vomiting are persistent **gastric lavage** may afford relief.

If the foregoing care is observed and the patient is not suffering from some acute systemic infection, especially of the respiratory tract, and if the condition of the heart and kidneys is satisfactory, the mortality from operation should be *nil*. Of 118 cases of acute appendicitis seen by the writer, 83 were operated with 2 deaths. Of the 35 acute cases not operated, none died. These were comparatively mild cases, in some of which there have been recurrences and the manifestation of chronic symptoms, while in others there has apparently been complete recovery.

CHRONIC APPENDICITIS.—Here again the only treatment that offers a reasonable prospect of cure is operation. Unfortunately many of these cases have already passed into a state of chronic invalidism and the obliterated appendix or the appendix bound down with adhesions is only an episode in their general ill health. Many of these improve with appendectomy only, but the great majority need additional care, dietary, orthopedic, mental and social. It is on account of the lack of appreciation of this both by the surgeon and by the patient, and on account of sometimes hurrying into the abdomen without proper consideration, that the surgery for chronic appendicitis is so frequently unsatisfactory.

Prognosis.—The mortality of appendicitis is yearly diminishing. This is because the diagnosis is made with greater confidence and certainty; the laity are in a receptive attitude and consent to an operation more readily; and the operation is done with greater safety. The acute cases make good recoveries unless certain complications already mentioned arise. The chronic cases are well, improved, or unsatisfactory, depending upon the chronicity of the disease and the gravity of the sequelæ which have arisen.

Pathology.—In undertaking a discussion of the pathology of appendicitis, acute or chronic, one must realize at once that it is impossible to offer a common pathological finding in explanation of the clinical findings and the clinical diagnosis (Klotz). Especially is this true of chronic appendicitis. In acute appendicitis the lesion follows more or less definite lines, such as are found in other like tissues. The mucous membrane of the appendix is studded with crypts very much the same as are found in the tonsillar tissue. These crypts may become infected and an inflammatory process, single or multiple, such as is seen in acute tonsillitis, may arise. When this process is confined to the mucosa it may be said to be a *simple* catarrhal process, when it extends to the submucosa it may be said to be *diffuse,* and if the inflammation proceeds to the pus-producing stage it is called *purulent* appendicitis. At either of these stages the tissues may maintain the power of repair, and after a time there may be no other evidence of the disease process having been present but localized scar tissue. In fact, the simple catarrhal or the diffuse forms may occur without any appreciable symptoms. It is not improbable that a localized purulent form may occasionally occur without recognized symptoms so long as the drainage of the particular crypt or crypts occurs, and so long as the lumen of the appendix is not encroached upon to interfere with adequate drainage. If, on the other hand, the inflammatory process continues to tissue death, or if drainage is interfered with, *gangrene* and *perforation* may ensue. Even at this stage the tissue defense may be so effective that repair may take place without surgical interference. It will be appreciated all the more, therefore, how difficult it is at times to differentiate between an acute attack in a more or less healthy appendix and an acute exacerbation in an appendix which already bears the evidence of previous invasion. There is no doubt, therefore, from a pathological standpoint, that acute appendicitis occurs much more frequently than surgical statistics may indicate. The clinician, no doubt, overlooks many attacks, or, even suspecting them, may refuse to make a diagnosis on the slight evidence at hand.

After many repetitions of acute catarrhal or acute diffuse attacks, the appendix takes upon itself the form of chronicity. In this condition, however, on account of interstitial scar tissue from ulceration, surrounding adhesions, retained fecoliths, or an obliterating process, any one of which conditions is liable to interfere with drainage, an acute exacerbation may occur which may be as definite in its course as though it occurred in a previously healthy appendix. The relative frequency of these conditions is shown by Klotz[24] in an analysis of 5,647 appendices examined. Of these 1,718 showed chronic interstitial lesions, 1,689 had adhesions, 832 were obliterated, and 195 had concretions.

When a localized or a general peritonitis occurs as the result of deep suppuration or of perforation, the consequent pathology may be in various locations and of various degrees. On account of the attachment of the mesentery and of other anatomical relations which cause the small intestine to gravitate towards the right iliac fossa a perforation may result in only a small portion of the peritoneum being affected. The condition is, as it were, "walled off" in a very small space where it may subside or form a localized abscess which may fortunately rupture into the bowel, or sometimes even through the external abdominal wall. Again the abscess, and this is the most frequent result, may rupture into the peritoneal cavity and produce a general peritonitis. If operation does not follow an attack, and localized peritonitis occurs with recovery, the appendix may be left attached or adherent to certain surrounding organs. Considerable portion of the organ, or only the tip, may be attached to the right tube and ovary, or even to the left, also to the bladder, to the cecum, to the ileum, to the duodenum, to the gall-bladder or to the stomach. Again, with a localized or a general peritonitis, a subphrenic abscess may develop or thrombosis and embolism may occur in various organs, especially in the liver or in the lungs. Pyemia may follow, of course, and be the ultimate cause of death.

Historical Summary.—Reginald Fitz's [25] paper on appendicitis was the beginning of a definite clinical and pathological understanding of the diseases of the appendix. Previous to this time the ultimate results of appendicitis were recognized as a typhlitis and a perityphlitis, but it was not known that the disease had its origin in the appendix. The only condition which occasionally attracted the attention of the profession to the appendix was that of foreign bodies. A paper by George Lewis of New York in 1856 was devoted entirely to a discussion of foreign bodies in the appendix. But this condition evidently was not looked upon as bearing a causal relation to typhlitis and perityphlitis.

The publication of Fitz's paper appeared just about the time when abdominal surgery became possible through the great work of Lister, Pasteur, and others. It is therefore to antiseptic surgery and to the definite statement that "perityphlitis, perityphlitic tumor, and perityphlitic abscess mean inflammation of the vermiform appendix," that humanity is indebted for the saving of lives and the relief of suffering from a disease which may have existed ever since the origin of man.

The literature of appendicitis since 1886 has dealt largely with the question as to whether appendicitis is strictly a surgical disease and as to the time and technic of operation.

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CHAPTER XXII

INTESTINAL OBSTRUCTION

By Horace W. Soper, M.D.
and
R. Walter Mills, M.D.[*]

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Definition.—Intestinal obstruction may be defined as an impediment to the onward passage of intestinal contents produced from any cause whatsoever, either intrinsic or extrinsic. The obstruction may be complete or incomplete, acute or chronic. An incomplete or chronic obstruction may suddenly become complete or acute. In the literature various terms are used in conjunction with intestinal obstruction.

Ileus is used to designate an obstruction due to paralytic conditions of the bowel.

Stricture or *stenosis* of the bowel is produced by a diseased condition of a limited segment of the gut resulting in organic contracture and narrowing of the lumen.

Constriction is a term employed to describe an obstruction produced by fibrous bands or adhesions outside the bowel.

By *compression* is meant an obstruction due to pressure by tumors, misplaced organs, etc.

Strangulation indicates that a loop of the intestine has been caught in such a manner as to shut off the circulation, producing venous stasis and gangrene.

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[*] Dr. Mills' participation in this article consisted in the preparation of the illustrations and in writing the section on "X-ray Evidence and Technic."

Occlusion is synonymous with complete obstruction.

Obturation is used when the obstruction is produced by material inside the bowel, such as coproliths, gall-stones, hair-balls, foreign bodies, etc.

Etiology.—Obstruction may be produced from a large number of conditions. Probably the most frequent is that due to bands of adhesions formed by previous inflammatory conditions. Meckel's diverticulum may produce occlusion in a similar manner. The tip of the appendix may become attached to other abdominal organs and act as an obstructing agent.

Stricture of the bowel may follow ulceration, tuberculous and syphilitic processes being the most frequent.

Foreign bodies such as gall-stones, intestinal concretions and parasites, fecal masses, etc., are more rare causes of obstruction.

Intussusception or invagination of the bowel is the most common cause in childhood and infancy.

Volvulus involving the sigmoid flexure or the coils of the small intestine is more frequent in persons of advanced years. Long-continued use of purgative drugs in chronic constipation may result in the formation of redundant loops of the colon. The sigmoid mesentery may become stretched and elongated.

Obstruction due to ileus or motor paralysis of the bowel may be secondary to toxic conditions affecting the neuromuscular apparatus. It may occur in typhoid fever, cholera, severe dysentery and has been observed in severe cases of pneumonia. However, the most frequent cause of such paralysis is *acute peritonitis.*

Embolism and thrombosis of the mesenteric vessels may occasion a paralysis of the affected portion and thus produce obstruction.

Carcinoma and new growths of the bowel as well as extrinsic tumor masses form a rather frequent cause of intestinal obstruction. Strangulation from internal herniæ is also a rather frequent occurrence. Among the most frequent are the retroperitoneal, retrocecal, intersigmoid, foramen of Winslow and diaphragmatic.

ACUTE OBSTRUCTION OF THE SMALL INTESTINE

Symptomatology.—Inasmuch as all forms of chronic obstruction may become suddenly acute, it would seem appropriate to consider primarily the symptomatology of acute obstruction.

As a rule the symptoms begin very abruptly. Intense pain, colicky in character, is the chief symptom. Vomiting is always present and is soon feculent. Collapse occurs early and is most intense when the obstruction is located high up in the small intestine. Stengel's * classic description of a patient in the stage of collapse cannot be too frequently quoted.

"The skin is cold, pale and bathed in sweat. The face wears an expression of anxiety and is pinched. The temples appear sunken. The eyes are depressed in their sockets and surrounded with dark rings. The respirations are shallow and often sighing. The temperature of the body falls. The pulse becomes rapid, feeble and finally thready and irregular. The mucous membrane becomes dry, so that the mouth is literally parched. Urinary excretion diminishes until it may become suppressed. Finally the patient sinks into a state of torpor or coma.''

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* STENGEL, ALFRED. Osler's Modern Medicine, 1908, VOL. V, 449.

The pain is usually referred to the middle of the abdomen, but exact localization possesses no real significance. It is important to note that in the early stage of acute obstruction the *abdomen* is *not sensitive* to pressure. It is only later when peritonitis develops that pressure tenderness is elicited. Distention of the abdomen occurs early and is progressive in character. Pain usually subsides as dissolution approaches. The sensorium is clear as a rule until near the end of the disease.

Constipation is absolute except that early in the illness several passages may occur. Enemas may wash out particles of fecal matter adhering to the colon.

Diagnosis.—Diagnosis is not difficult in typical cases. Indicanuria is frequently referred to as a diagnostic sign, but is in reality unreliable inasmuch as many persons in apparently good health have habitually a high indican content. It is true, however, that a strong indican reaction is present in nearly every case of acute obstruction. Therefore, a negative indican reaction would militate against the diagnosis of intestinal obstruction in every obscure acute abdominal condition.

A number of intra-abdominal conditions may simulate acute intestinal obstruction, *e. g.*, biliary and renal colic, perforating gastric and duodenal ulcers, acute perforating appendicitis, Dietl's crisis, acute pancreatitis, embolism or thrombosis of the mesenteric vessels. In all of the above-mentioned conditions the clinical picture of sudden intense pain, vomiting and collapse is present. The abdominal distention, however, is not so marked as in intestinal obstruction. The vomitus is rarely feculent. In biliary and renal colic and Dietl's crisis, the localization of the pain usually suffices to differentiate. Moreover a hypodermic of morphia will usually produce an amelioration of the gastro-intestinal symptoms. In acute obstruction, however, the morphia may temporarily lessen the severity of the pain, but *will in no degree check the progressive abdominal distention, eventual fecal vomiting and collapse.*

In *perforating* gastric and duodenal ulcer and appendix the leukocyte count is high. The chief point of *differentiation*, however, is the presence of *involuntary rigidity of the abdominal muscles,* usually limited to the right side. Involuntary muscular rigidity is not present in acute intestinal obstruction unless the parietal peritoneum is involved.

In embolism and thrombosis of the mesenteric vessels the hemorrhagic character of the vomitus and stools are differential points; moreover a palpable mass may be present and usually the presence of endocarditis is demonstrable.

Acute hemorrhagic pancreatitis usually produces a distention in the epigastrium. Early muscular rigidity is present, due to the early development of localized peritonitis.

Finally in the consideration of the above-mentioned conditions, such as perforative peritonitis, actue pancreatitis, embolism of the mesenteric vessels, all of which may simulate acute intestinal obstruction, we must emphasize that all are diseases that **demand immediate surgical intervention.** No time should be wasted on differential diagnostic tests. The diagnosis of "acute abdomen" as it has recently been termed, suffices. Surgical relief must be prompt. Early operation will save many lives. Late operation is futile and serves only to bring the method into disrepute.

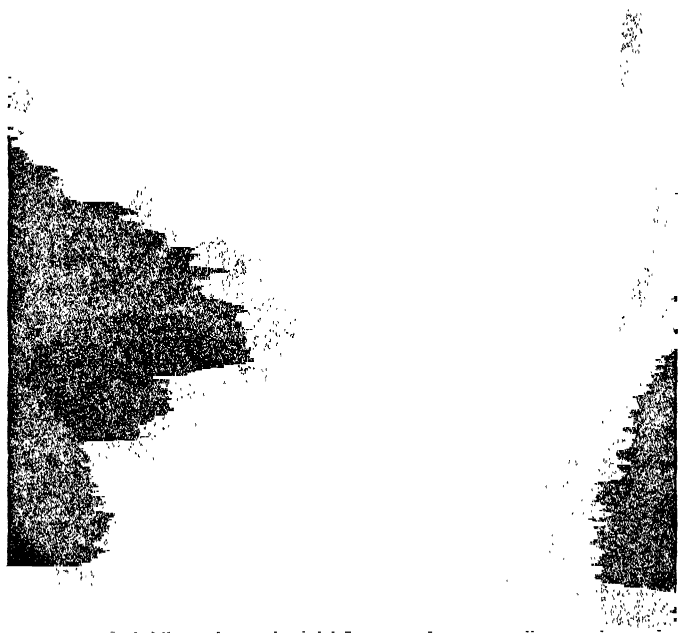


FIG. 1.—MARKED REDUNDANCY OF ILEAC, DESCENDING AND TRANSVERSE COLONS. GENERALLY CONSIDERED A CONGENITAL ANOMALY BUT A CONCOMITANT OF MARKED COLONIC STASIS AND QUESTIONABLE IN THE AUTHORS' OPINION AS TO WHETHER, AT LEAST IN ITS LESSER MANIFESTATIONS, A RESULT AND NOT A CAUSE.

ACUTE OBSTRUCTION OF THE LARGE INTESTINE

Etiology.—Volvulus of the sigmoid loop is the chief cause of acute obstruction in the large intestine. Redundant loops frequently occur in the pelvic colon, hepatic flexure and near the splenic flexure in individuals near or after middle age. Mills has made a special study of a large number of cases of this character and is of the opinion that chronic constipation and the habitual use of cathartics are etiologic factors in the formation of these redundancies; in other words, they are acquired and not congenital.

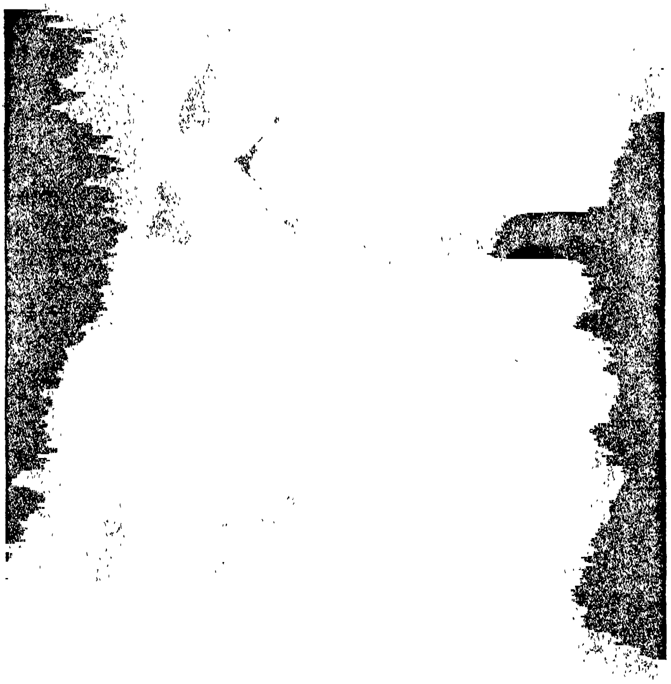


FIG. 2.—ANOTHER CASE OF REDUNDANCY HERE OF ILEAC AND PROXIMAL TRANSVERSE COLONS. (See legend of preceding illustration.)

INTUSSUSCEPTION.—Intussusception is a frequent cause of acute obstruction occurring in children. It usually involves the ileum and cecum or the lower coils of the ileum, but may occur in any part of the intestinal tract. The onset is slow as a rule but may be very sudden in young infants. Palpation of a sausage-like tumor, bloody stools and the symptoms of intestinal obstruction usually establish the diagnosis.

Enemata of emulsified oil are especially to be advised in intussusception. Care must be exercised and not too much force applied. The solution should be allowed to flow in slowly (Figs. 1 and 2).

Symptomatology.—The symptoms are practically the same as in obstruction of the small intestine except that they are slower in development. The vomitus rarely becomes feculent and the onset of collapse is not so rapid. The pain is usually localized in the early stages. Visible peristalsis is usually observed. The distended loop may often be palpated.

Pathology.—In the purely acute cases there is no evidence of hypertrophy of the bowel. Below the point of obstruction the bowel is empty and contracted; above the obstruction the bowel is greatly distended and the walls very thin. In the beginning the contents of the bowel are liquid or semifluid, but after a short time large accumulations of gas occur partly as a result of interference to the circulation as well as the inability of the mucous membrane to absorb the intestinal contents. Moreover a portion of the gaseous accumulation may be produced by the carbon dioxid derived from the venous blood of the stagnant intestinal circulation. When the obstruction is produced by strangulation the involved loop of the bowel quickly becomes gangrenous. Localized peritonitis frequently occurs about these strangulated areas. The wall of the bowel above the obstructed area undergoes rapid degenerative changes and allows the passage of infectious agents into the surrounding peritoneum, causing localized and eventually general peritonitis.

CHRONIC INTESTINAL OBSTRUCTION

Symptomatology and Diagnosis.—The symptomatology of chronic obstruction is practically the same in all cases regardless of the etiologic factors involved. Special characteristic symptoms will be considered under the different causes of obstruction.

Intermittent colicky pain should arouse the suspicion of an obstructive agent. Constipation is usually present, but may be absent. Diarrhea sometimes occurs, but as a rule the passages, while numerous, are upon investigation found to be inadequate.

Visible peristaltic movements are of extreme importance in diagnosis. During the attack of pain the bowel above the obstruction becomes distended into a mass of almost tumor-like hardness. It finally relaxes with relief from the pains until another paroxysm occurs. This *intermittent spasm of the intestine is an extremely important sign of obstruction.*

In every case of chronic obstruction of the bowel there is, as a rule, a history of digestive disturbances. The patient is conscious of gaseous distention or a vague feeling of general abdominal discomfort. A gradually developing constipation in a patient who, as a rule, has had good bowel evacuations should arouse the suspicion of colonic obstruction due to organic disease.

Pathology.—The wall of the bowel above the site of obstruction is hypertrophied. The hypertrophy affects chiefly the musculature and is compensatory in character. The mucous membrane presents inflammatory changes. Frequently ulceration involving the submucosa occurs.

The serous coat is stretched and thinner than normal. Below the point of obstruction the bowel is collapsed and empty.

CARCINOMA

Small Intestine.—Carcinoma of the *small intestine* is relatively rare. Because of the fluid contents of the intestine the growth is far advanced before obstruction occurs.

FIG. 3.—SMALL INTESTINAL OBSTRUCTION RESULTING FROM CARCINOMA. TWO OR THREE CARCINOMATOUS NODULES INVOLVING THE WALL OF THE SMALL INTESTINE AT THE INSERTION OF ITS MESENTERY, RESULTING IN RATHER MARKED LOCAL OBSTRUCTION. OPERATIVE CONFIRMATION.

Colicky pains, rapid weight loss and persistence of occult blood in the feces on flesh-free diet are the chief diagnostic points. Roentgen ray examination may give no clue unless the obstruction is almost complete. The annular type, located in the ileum, may give no symptom whatsoever until occlusion occurs; the syndrome of acute obstruction then appears (Fig. No. 3).

Carcinoma of the jejunum and ileum may progress to fatal issue before obstruction of the bowel is produced. The hope of an early diagnosis in carcinoma of the small intestine is based upon the systematic examination of the feces for occult blood. Ulceration occurs in the vast majority of these growths. Therefore, every case presenting progressive loss of weight and strength without obvious cause, particularly if associated with colicky pains or irregularities in the fecal evacuations, should be subjected to a careful examination of the stools for occult blood. The occult blood test, done occasionally and in a careless manner, often leads to erroneous conclusions and unfortunate results. However, when performed carefully and systematically, with due regard to all sources of errors, it is the most valuable of all the objective methods at our command in the establishment of the diagnosis of ulcerative lesions in the gastro-intestinal tract. The benzidin and phenolphthalein tests are really too delicate to be relied upon. The *guaiac test* is by far the most reliable. The technic is as follows:

A portion of the feces, about the size of a walnut, should be selected, avoiding surfaces covered with mucus, mixed with water and rubbed well in a mortar to a thin mushy consistency. If the stool is liquid, the sediment should be selected without the addition of water. Pour from 5 to 10 c.c. in a large test-tube, add one-third volume of glacial acetic acid, mix well, add equal volume of ether and extract slowly. Use a rubber cork and tilt the tube in order to bring the largest possible surface of the fluid into contact with the ether without permitting them to be mixed together. Should a mixture, however, occur, the resulting emulsion may be filtered and the filtrate used. At least three minutes' time is required to complete the extraction. The ethereal extract is carefully poured off into a second test-tube and from 10 to 15 drops of freshly prepared tincture of guaiac and from 20 to 30 drops of old oil of turpentine added. Shake well in the air without working or covering with the thumb. The appearance of a blue or violet color indicates the presence of blood. Should the color not appear at once, fill the test-tube two-thirds full of water. The ethereal extract will float on the surface and the color change is more readily detected. Other colors may appear, but, after much discussion, observers are practically unanimous in advising *that only a blue or violet color should be accepted as a positive reaction.* The color is not lasting, but fades and disappears in a few minutes.

Meat, including fowl and fish, and all meat soups and extracts must be excluded from the dietary for a period of three days before the test is made. All possible sources of accidental bleeding must be kept in mind, *e. g.*, gums, throat, nose, etc. In females the menstrual period must be avoided.

Large Intestine.—Carcinoma of the large intestine is a frequent cause of obstruction. Painless constipation may be the only symptom for a long period. The observant patient will, however, note the presence of blood in the feces. This observation should always lead to exhaustive examination, which should not cease until the cause of the bleeding is definitely ascertained. As Nothnagel emphasizes, the admixture of pus with blood is indicative of carcinoma; hemorrhoidal blood is usually free from pus. In carcinoma the blood predominates; in pus proctitis the pus content is larger than the blood and a more intimate mixture occurs. In all cases of colonic bleeding the blood is bright or dark red in color, never black. Blood from the cecum is usually dark red and clotted, while blood from the lower colon is bright red in color.

Fig. 4.—Marked Obstruction Median Transverse Colon Due to Carcinoma (?). Proximal Stasis after Barium Meal (see following figure).

We must note, however, that the annular type of carcinoma may cause a constriction of the bowel without producing ulceration of the mucosa; in this event no blood will appear in the feces. Sigmoidoscopy should never be neglected and will often establish the diagnosis without further examination. An extremely important point in sigmoidoscopy is the observance of flecks of blood adhering to the mucosa of the bowel. Fecal masses passing through an ulcerated area will often drag masses of blood along the colonic wall leaving a "trail" that is strongly suggestive of an ulcerated growth higher up in the bowel beyond the reach of the sigmoidoscope.

Weight loss is *not* an early symptom of cancer of the colon, but always a sign that the growth is far advanced. Therefore, one should not look for it in the consideration of growths involving the large intestine, particularly the lower half. On the contrary early weight loss is always to be considered in the diagnosis of cancer of the *small intestine*.

FIG. 5.—OBSTRUCTION MEDIAN TRANSVERSE COLON AS VISUALIZED BY BARIUM ENEMA. (Same case as preceding figure.)

Careful repeated *palpation* may at times establish the diagnosis of carcinoma when all other methods fail. In one of the author's cases the growth had involved the wall of the upper pelvic colon in so symmetrical a fashion that the x-ray plate gave no evidence of its presence. The diagnosis, confirmed by surgical operation was made solely on the palpatory findings (Figs. 4, 5, 6, 7 and 8).

X-RAY EVIDENCE AND TECHNIC

Because of the great value of the roentgen ray in the diagnosis of intestinal obstruction, the authors will describe here in detail the technic they employ in x-ray examination of the intestinal tract, as it relates to obstructive conditions.

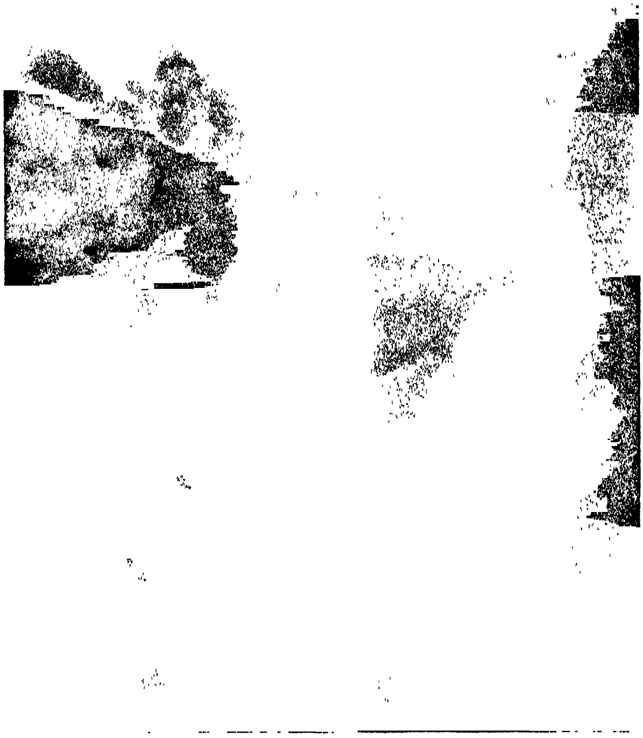


Fig. 6.—Carcinoma Involving Ileac Colon, Resulting in Quite Marked Obstruction. Plate after Barium Enema Indicating Effective Manner in Which Enema Outlines Entire Obstructive Factor. Operative Confirmation.

General.—The value of the x-ray in the diagnosis of intestinal obstruction is great but it must be emphasized that evidence by screen or plate should be subjected to most careful interrogation, if at all possible, by one of large experience. So many artefacts and suggestions of obstruction or obstructive dilatation occur, which are in reality not the result of obstruction, that conclusions should be drawn with the greatest caution. It may also be emphasized that x-ray indications should be well controlled with clinical findings and best interpretated in close relationship to such.

The x-ray as an aid in eliciting facts of diagnostic value is useful in the determination of the existence of intestinal obstruction, and, within some limits, its location and, in less measure, its nature. Localization is much more accurate, in fact usually exact, in the colon while less frequently so in the small intestine with the exception of the terminal ileum. The nature of the obstruction is never to be positively decided by the x-ray, though the degree of probability as to the precise nature of the obstruction is in a large number of cases considerable. Again accuracy in this regard is much greater in obstructive lesions of the colon as in the instance of carcinoma, pericolitis resulting from diverticulitis and in stricture of the distal colon as from postluetic contractures than in small intestinal obstructive conditions. It is a curious paradox that the most valuable information derived by the x-ray in

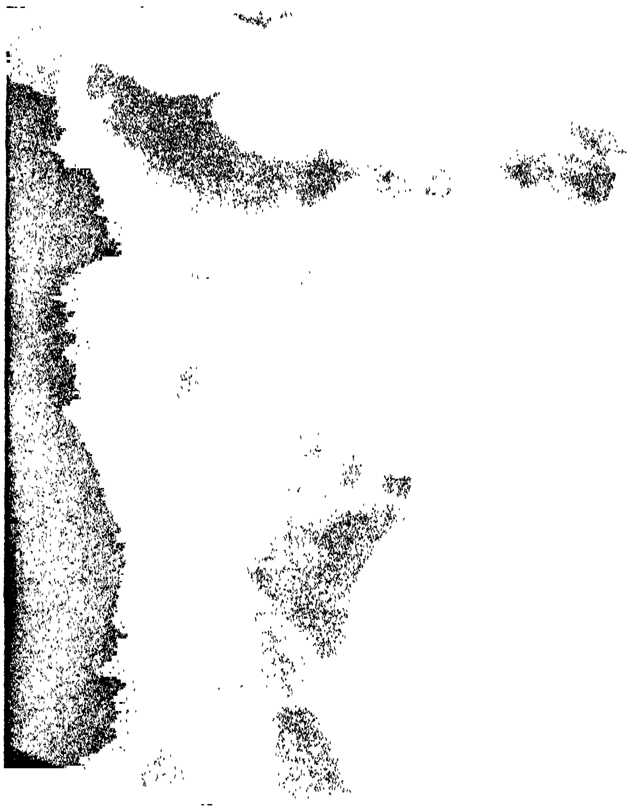


FIG. 7.—CARCINOMA OF CECUM AND PROXIMAL ASCENDING COLON RESULTING IN DEGREE OF OBSTRUCTION. BARIUM ENEMA. OPERATIVE CONFIRMATION.

obstructions of the intestines is on the whole not obtained through evidence of delayed motility but through the form of the gut proximal to the obstructive point as indicative of dilatation, in less measure hyperperistalsis and dislocation. Many functional delays in small intestinal motility occur and more confusingly delay in proximal motility as from marked slowing in the time of stomach emptying may give a misleading secondary delay in the small intestines. A decided gastric functional motor insufficiency, as occurring in a patient nauseated or suffering from severe headache or after the eating of ordinary food against orders in the next few hours following ingestion of a barium meal, may result in such marked delay in the evacuation of the stomach contents that the small intestine will be found barium-filled after its final expulsion from the stomach hours after the usual time. In functional colonic stasis,

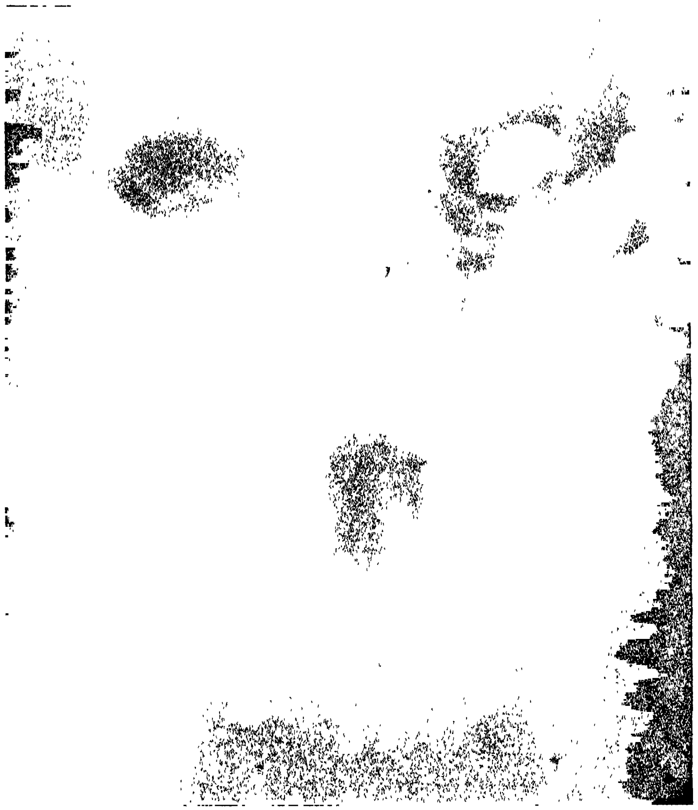


FIG. 8.—COLONIC OBSTRUCTION, RESULT OF SMALL ANNULAR CARCINOMA OF MEDIAN TRANSVERSE COLON. CHARACTERISTIC COLONIC OUTLINE INDICATING SLIGHT DILATATION THROUGH DEGREE OF STASIS SEEN PROXIMAL TO OBSTRUCTIVE LESION. OPERATIVE CONFIRMATION.

progression of the bariumized portion of the colonic contents may be greatly delayed with some degree of dilatation of the colon present, yet such neither indicate nor localize actual obstruction but be due to an unbariumized fecal column ahead not to be visualized by the x-ray. In the colon there is frequently on surface indications marked barium retention proximal to an obstructive point to a greater degree than is actually the case, the retention and superfilling of the colon proximal to the obstruction being the result of liquefaction of its contents. Such a picture may be obtained in spite of a history of numerous diarrheic defecations and without clinical indications of a considerable obstruction. On the other hand the form of the intestine as expressed by varying degrees of dilatation and hyperperistaltic response proximal to an obstructive point offer pretty definite criteria as to the presence and degree of an obstruction.

There are three ways of elicitating x-ray evidence of intestinal obstruction together with their modifications and combinations: First, the ordinary fed test per orem; second, the open plate method; and third, the barium enema.

The Ordinary Fed Test Per Orem.—The question of the advisability of the administration of a barium meal to a patient with any degree of intestinal obstruction must be considered. In cases of moderate obstruction this may be done with impunity. The authors have never had ill results even where a barium meal was given inadvertently in case where marked obstruction was present. However, it is wiser if there be a possibility of a definite degree of obstruction being present to use one-half the amount of the barium meal or even less and a smaller amount of barium than the usual percentage in that portion of the meal.

The Open Plate Method.—The taking of a plate of a patient with an intestinal obstruction without the administration of barium in any form or way is of the greatest practical value, especially in cases of marked and acute obstruction. The obstructed small intestine of characteristic outline, dilated, gas- and fluid-filled will be shown and at least an approximated localization of the obstructed point afforded. If the patient is too ill to allow of taking a plate in the prone position, it may be taken while recumbent. If possible a plate with the subject standing should be made, as evidence of fluid levels so to be obtained is often of value in determining the presence and locality of the obstruction. This method of plating without contrast substance is less applicable, helpful and necessary in the instance of colonic obstruction; still its worth is scarcely impaired by this fact, as cases of colonic obstruction are not usually so urgent but that other methods of x-ray examination may be attempted, notably the administration of a barium enema which will practically always determine at least the distal boundary of the area of obstruction.

The Barium Enema.—This means of extreme value is, as mentioned, always available in determining the existence, position and, usually with a fair degree of accuracy, the nature of a colonic obstruction. It serves as a valuable means of elimination in cases in which obstruction is suspected but not present in the colon, frequently inclusive of the terminal ileum, which may be shown to be not obstructed by a reflux of the enema through the ileocecal valve. If the degree of obstruction is known not to be great on clinical grounds, a preparatory laxative in divided doses may be given. A barium enema may with care be given in the case of a very sick patient. The authors have used it in subjects practically moribund for the determination of the obstructive point. It is unnecessary to attempt any sort of preparation, as the colon distal to the obstruction is usually practically empty and even if it be not, with gentle coaxing the enema may be made to fill the colon about the central fecal matter and an adequate outline so obtained. It is curious but true that the barium enema affords better criteria of the presence and degree of an obstruction than does the fed test. Owing to responsive hypertrophy of the colon proximal to the obstructive point, proximal motility may not be greatly impaired. On the other hand in the same case efforts to drive a retrogressive barium enema beyond the obstructive point will meet with failure, a fact most valuable in its diagnostic significance. The presence of intestinal gas alone is often of diagnostic usefulness, especially in the colon and particularly in the distal colon, as an indication of the position of an obstructive point. Its interpretation should be attempted with extreme caution. In the small intestines gas may aid in a general way in determining what portion is obstructed, but as considerable collections of gas occur in the hypotonic small intestine in debilitated states without an obstruction or precise pathological condition being present, the indication should also be used with caution. At times the use of a saline laxative after a fed test in cases where obstruction is known not to be very marked is helpful in liquefying the bariumized contents of the intestine proximal to the obstructive point and so developing facts pertaining to its dilatation, hyperperistalsis and point of obstruction.

It is true that the x-ray plate is much more helpful in obtaining information regarding obstruction than is the roentgenoscopic screen. The latter lacks detail and the opportunity for deliberate study available by the former; still both methods are best employed. The screen often gives directional information helpful in obtaining plates and is at times otherwise useful. The screen is of great aid in cases of intestinal obstruction through the opportunity which it affords of combining palpation with roentgenoscopy to the determination of palpable findings as registering with presumable points of obstruction, and so affording either corroborative evidence or sometimes equally valuable, failure to do so. The determination of a palpable finding as registering with a point of obstruction occurs very much more frequently in the instance of the colon than in that of the small intestine. It not infrequently happens that small tumors which would escape detection without the information thus leading to their localization become definitely palpable at the point of obstruction. When occurring deep in the pelvis such tumors often cannot be palpated, even with roentgenoscopic aid, except on the administration of a barium enema causing the distal colon to rise out of the pelvis to a degree. Another helpful aid afforded by roentgenoscopy is the determination of a point of suggestive tenderness as registering with a suspected point of obstruction, corroborating the localization. Such evidence is to be interpreted with care where peritoneal inflammation, not necessarily associated with obstruction, is present, as in the ileocecal region. Immobility on palpation of the part involved in an intestinal obstruction is at times of help, though personally the authors cannot consider it a point of value equal to certain of those mentioned.

X-ray Evidence of Small Intestinal Obstruction.—The particular direct evidence indicating small intestinal obstruction is apparent on the plate and in much less measure roentgenoscopically, by intestinal outlines indicating dilatation and a peculiar herringbone or ribbed pattern resulting from the presence of the *valvulæ conniventes.* It is helpful that these markings attributable to the presence of the normal folds of the small intestinal mucosa are not developed in nonobstructive cases; as their differentiation on the plate seems dependent upon a certain thinning of the gut only resulting through a greater degree of dilatation than occurs in conditions of nonobstructive distention, as from small intestinal hypotonicity. The fact that the *valvulæ conniventes* decrease in number and degree of development from the second portion of the jejunum to the terminal ileum, in which they are absent, is a helpful factor in the general localization of the point of the obstruction.

The degree to which these abnormal forms indicating obstruction are apparent on the plate varies naturally with the degree of obstruction and resulting dilatation through a wide range from spectacular pictures positively recognizable at a glance to less marked, questionable and doubtful indications fading into the realm of uncertainty. Multiple obstructions are frequently evinced by local puddlings with fluid and gas levels at various points throughout the area of the small intestine. The nature of the obstructive factor is not revealed by x-ray evidence in the small intestine other than as to probabilities. For instance, small intestinal obstruction in the region of the ileocecal mechanism without palpable findings naturally leads to the presumption of its occurrence through an inflammatory exudate secondary to appendicitis, ileal kink, and so on. A local palpable mass would suggest the presence of small intestinal carcinoma. Palpable findings indicating a general peritoneal process or neoplasm lead to the presumption of stenosis, or stenoses, attributable to such.

X-ray Evidence of Colonic Obstruction.—Certain statements have been made regarding x-ray evidence of intestinal obstruction in a general way as to the technic of its elicitation and pertaining to its interpretation that apply to the subject as regards the colon in particular. Thus it may be repeated that certain decisive evidence results from changes in form and, in less measure, in position of the colon proximal to the point of obstruction as well as from actual delay in the progression of the barium meal, and that evidence of the latter condition while of great help should be interpreted with caution; that best information is often derived through the use of barium enema and its failure to progress; and that the use of laxatives in liquefying the content of the colon to the determination of the point of obstruction is especially useful in the colon. An observation after a combined fed test and barium enema, or after the combination of a fed test, liquefaction of feces through a laxative, and a barium enema is especially helpful in the instance of the colon. An opinion as to the degree of obstruction is a matter of judgment compounded through the consideration of all data obtained, and, again repeating, should be carefully correlated with clinical findings. In turn the necessity of a summation of the various evidences obtained as the result of the x-ray examination alone, such as registration of an obstructive point with palpable findings and suggestive tenderness, must be recalled.

Certain evidence previously mentioned is especially applicable in the instance of colonic obstruction as compared to that of the small intestine. Thus accumulation of gas proximal to the obstruction point is much more definite and valuable in the instance of the colon. The presence of hyperperistalsis is a useful and often an exact means of determining the presence and locality of an obstruction in the colon, provided a laxative has not been taken; however, this is not true in the instance of the small intestine. Care in interpretation is especially necessary in considering the signs of colonic obstruction, more so than in the instance of the small intestine, because the diagnostic deductions may be carried further in the instance of the colon and consequently must be more carefully considered. Pressure artefacts of the colon, as by the spine, extra-alimentary tumor and the like, hyperperistalsis resulting from the action of laxatives often taken without the examiner's knowledge, and collections of gas due to colonic hypotonicity give pseudo defects which require conservative interpretation.

The various lesions in the colon producing colonic obstruction so give evidence not only of their presence and position, but in a considerable measure a probability as to their nature. Carcinoma of the colon gives a contour defect variable and characteristic in its different locations. The same applies in scarcely less measure in the instance of those exudative reactions secondary to colonic lues. Evidence of colonic strangulation as by adhesions, herniæ, motor paralyses as from general peritonitis and the like may be suggested by methods reviewed, especially by the plate taken without barium ingestion or clysma. Obstruction by tuberculous exudate, as in the instance of tuberculous tumors of the cecum, gives a picture of obstruction and a contour defect not to be differentiated from that of carcinoma. Local colitis, especially the secondary results of ulcerative colitis, gives a picture of usually moderate obstruction similar to that occurring in postluetic conditions. Pressure, as from extra-alimentary tumors, particularly if the colon be secondarily involved, not infrequently results in a moderate degree of obstruction, though such involvement may lead to a confusing nonobstructive inhibition of colonic motility, both proximal and distal. Usually, however, differential points, especially the lack of an inherent filling defect and the presence of local colonic outlines suggesting extrinsic pressure, are available. Obstruction following peridiverticulitis is one of the common causes of colonic obstruction, usually moderate in degree.

DIVERTICULOSIS OF THE COLON

Diverticulosis of the colon may occasion partial or even complete obstruction. In acute diverticulosis the pus sac may be so large as to temporarily obstruct the lumen. The fever, localization of the pain, tenderness and finally pus in the feces make the diagnosis obvious in the typical cases. Wm. H. Mayo has demonstrated that chronic diverticulitis may simulate carcinoma so closely that differentiation is impossible even on the operating table. Moreover, he has shown that carcinoma is especially likely to develop on the basis of chronic diverticulitis.

FIG. 9.—MODERATELY MARKED COLONIC OBSTRUCTION PELVIC COLON, RESULT OF CARCINOMA ASSOCIATED WITH AND PROBABLY SECONDARY TO DIVERTICULITIS. OPERATIVE CONFIRMATION.

In carcinoma the presence of blood in the feces will almost invariably occur. In diverticulosis it is absent. Therefore a patient suffering from chronic diverticulitis should be kept under observation; if blood appears in the feces malignancy should be strongly suspected (Fig. 9).

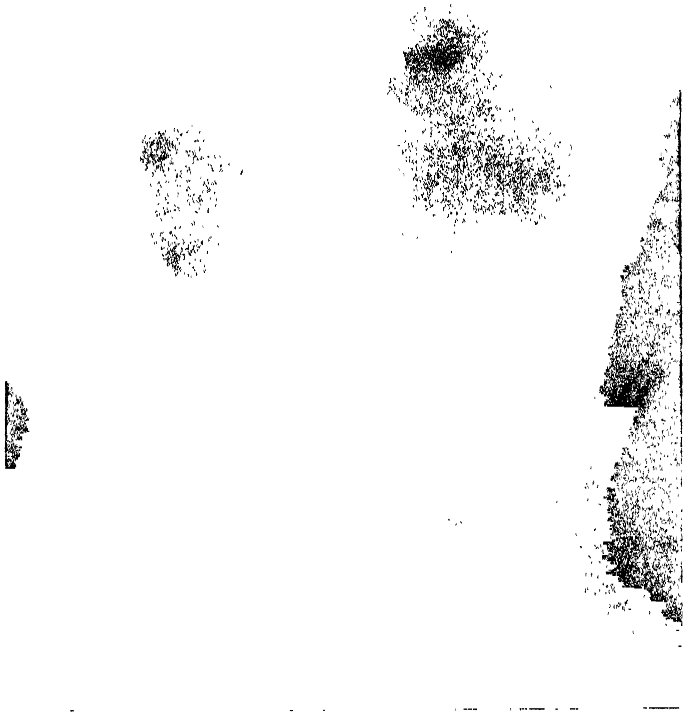


FIG. 10.—OBSTRUCTIVE CONTRACTURE PROXIMAL RECTUM AND PROBABLY PELVIC COLON, RESULT OF SECONDARY CONTRACTURE DUE TO LUES. OPERATIVE CONFIRMATION.

CICATRICIAL STRICTURES

The consideration of strictures shall be limited to those formed by ulceration of the bowel. The chief causes are tuberculosis, stercoral accumulations, syphilis and dysentery; stricture following typhoid fever is exceedingly rare.

Dysenteric ulcers were formerly regarded as a frequent cause of stricture. As a matter of fact but few authentic cases appear in the literature. Syphilitic ulcer, common in the rectum, is a rare finding in the colon or small intestine. The authors have observed it in several cases involving the pelvic colon (Fig. 10).

Tuberculous stricture may be single or multiple. The tuberculous ulceration is deep, runs transverse to the long axis of the bowel, is frequently attended with hyperplastic processes that subsequently give rise to connective tissue formation.

Annular contractions develop which may cause partial obstruction in both small and large intestine. Complete obstruction rarely, if ever, is produced by tuberculous ulceration.

FIG. 11.—MULTIPLE OBSTRUCTIONS SMALL INTESTINE RESULTING FROM TUBERCULOUS PERITONITIS. CHARACTERISTIC PATCHY IRREGULAR PUDDLING. OPERATIVE CONFIRMATION.

In chronic tuberculous peritonitis, as well as in carcinomatosis, the motility of the small intestine is disturbed in a characteristic manner. Bands and adhesive processes may be formed which occasion the onset of acute obstruction (Fig. 11).

Compression of the bowel is usually produced by the pressure of tumors. Abscesses may occasion obstruction; it is, however, as a rule, only partial. The fixed portions of the large intestine are relatively more frequently involved than the small intestine. The adaptability of the small intestine and its escape from compression agencies is remarkable (Figs. 12 and 13).

OBSTRUCTION DUE TO MOTOR PARALYSIS OF THE BOWEL

Obstruction due to motor paralysis may be partial or complete. It may occur in conditions not affecting the bowel itself, *e. g.*, injuries of the testicle and ovary, and inflammatory conditions in the inguinal regions after pelvic and hemorrhoidal operations, and in renal and biliary colic. In any of these conditions a functional paralysis of the intestine occurs without any evidence of organic change in the wall of the bowel. According to Meltzer's theory of antagonistic innervation,

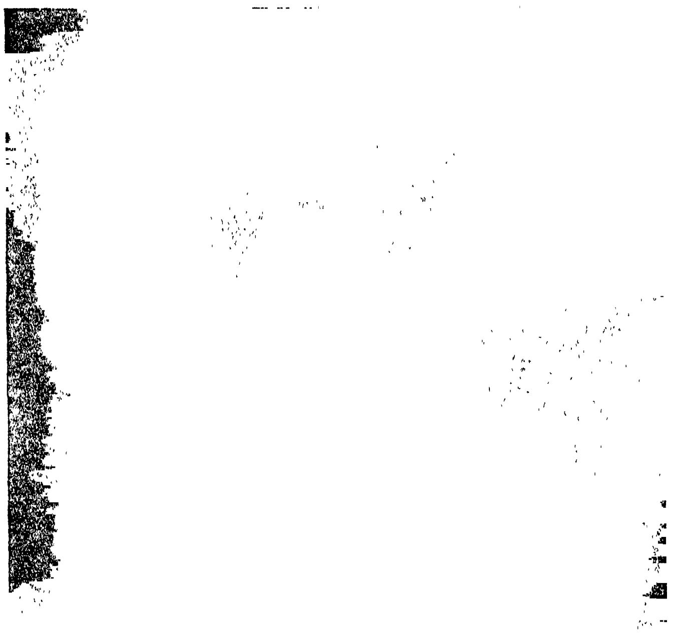


FIG. 12.—MARKED COLONIC STASIS DUE TO OBSTRUCTION IN DISTAL TRANSVERSE COLON RESULTING FROM EXTRA-ALIMENTARY TUMOR PRESSURE, RENAL NEOPLASM (?). (Operative confirmation but origin of tumor not determined.)

this paralysis would be explained by the irritation or overstimulation of the inhibitory neuromuscular apparatus.

Toxic conditions occurring in the course of pneumonia, typhoid fever, cholera, etc., may act upon the neuromuscular apparatus in the same manner as the above-mentioned direct agencies, and produce a functional paralysis and consequent obstruction.

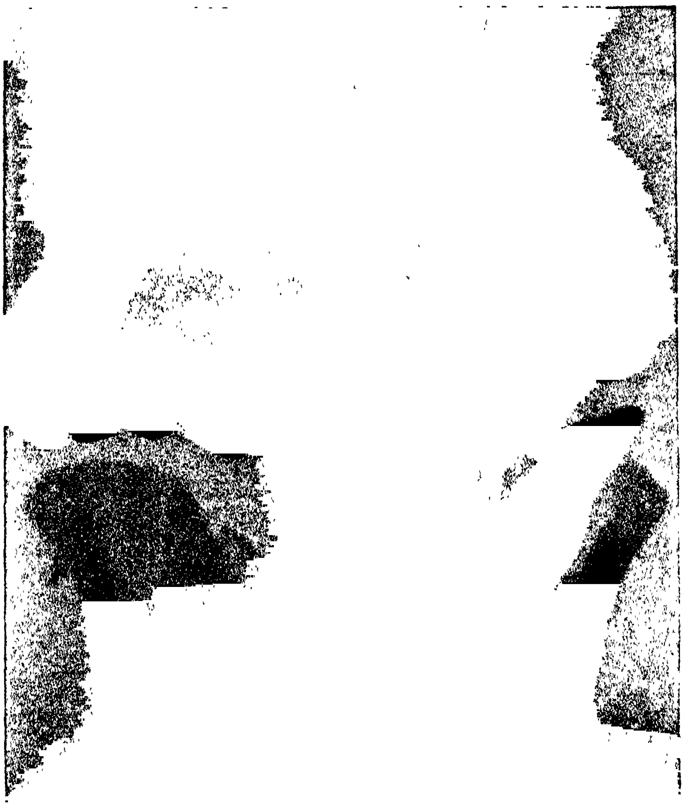


FIG. 13.—COLONIC OBSTRUCTION RESULTING FROM EXTRA-ALIMENTARY TUMOR PRESSURE AND INVOLVEMENT OF TRANSVERSE COLON. (Same case as preceding. Plate after barium enema.)

Paralysis of the bowel in organic disease is more common. In acute peritonitis complete obstruction quickly results. In the various forms of local peritonitis localized segments are involved. In cases of this character the resulting paralytic condition of the bowel is caused by direct involvement of the wall of the bowel in the infectious process as well as the effect of this condition on the nervous apparatus that controls peristalsis (Fig. 13A).

OBSTRUCTION DUE TO FOREIGN BODIES

Obstruction due to gall-stones may occur in any part of the small intestine; the most common location is in the lower ileum. The symptoms are those of acute obstruction. Fruit stones, masses of intestinal parasites, hair-balls, masses of bismuth, salol and chalk may form the nucleus for an obstructive mass, the fecal matter accumulating about the foreign body.

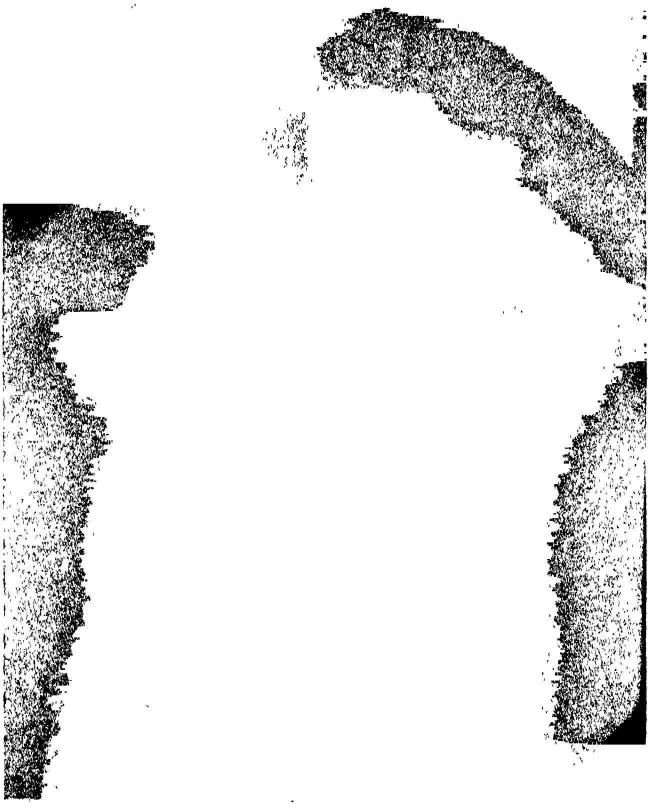


FIG. 13A.—MARKED SMALL INTESTINAL OBSTRUCTION AS THE RESULT OF ILEUS SECONDARY TO EXTENSIVE MILIARY ABDOMINAL POSTOPERATIVE CARCINOMATOSIS, PRIMARY LESION IN ILEAC COLON. PLATE SHOWS FLUID LEVELS RESULT OF MARKED STASIS IN SMALL INTESTINE. PLATE TAKEN WHILE STANDING. OPERATIVE CONFIRMATION.

Intestinal concretions *per se* may cause partial or complete obstruction of the colon. The fecal mass may reach an enormous size. At times it is channeled in such a manner that diarrhea is produced. In cases of this character the symptoms develop slowly; in fact the patient may be so free from discomfort that the mass is discovered during a routine examination. A patient who came under the authors' observation suffered from an ulcer involving the anal sphincter to such a degree that he refrained from defecation for a period of six months' time. The entire colon was filled with a hard fecal mass which required surgical removal. Yet he presented no symptoms of obstruction and reluctantly consented to the operation. His urine showed only a slight trace of indican. This case is of interest in illustrating the degree of tolerance of some individual colons for the retention of fecal masses. As a rule the patient develops symptoms, such as headache, vertigo, nausea, anorexia and lassitude. If the mass forms in the cecum, symptoms of acute obstruction may finally occur.

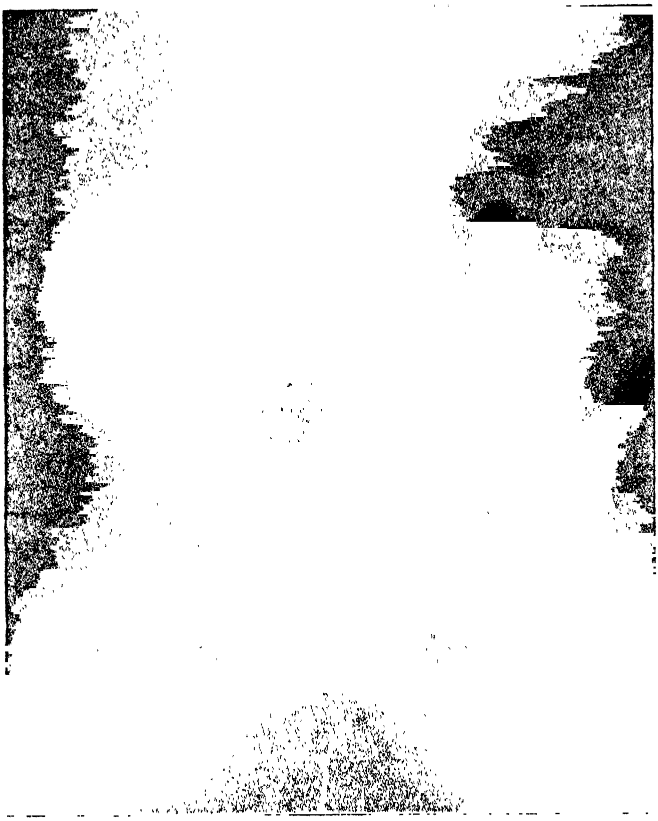


FIG. 14.—PROFOUND COLONIC STASIS SECONDARY TO PURULENT DISTAL COLITIS AND DOUBTLESS PROTECTIVE INHIBITION. PLATE SEVEN DAYS AFTER INGESTION OF BARIUM. NO PROGRESSION AT ANY TIME BEYOND SPLENIC FLEXURE. THREE DAYS LATER AFTER A LAXATIVE ENTIRE BARIUM EVACUATED INDICATING NO ORGANIC OBSTRUCTION.

OBSTRUCTION DUE TO CONSTIPATION

Chronic constipation produces partial obstruction to the fecal current. It is often severe enough to affect the motility of the entire digestive apparatus. Fecal stasis develops in the redundant atonic colonic loops. Stasis in the colon may be caused by inflammatory conditions in the rectum and lower pelvic colon. One of the authors' cases of pus infection in the rectum and pelvic colon showed a fecal retention in the transverse colon of one week's duration (Fig. 14).

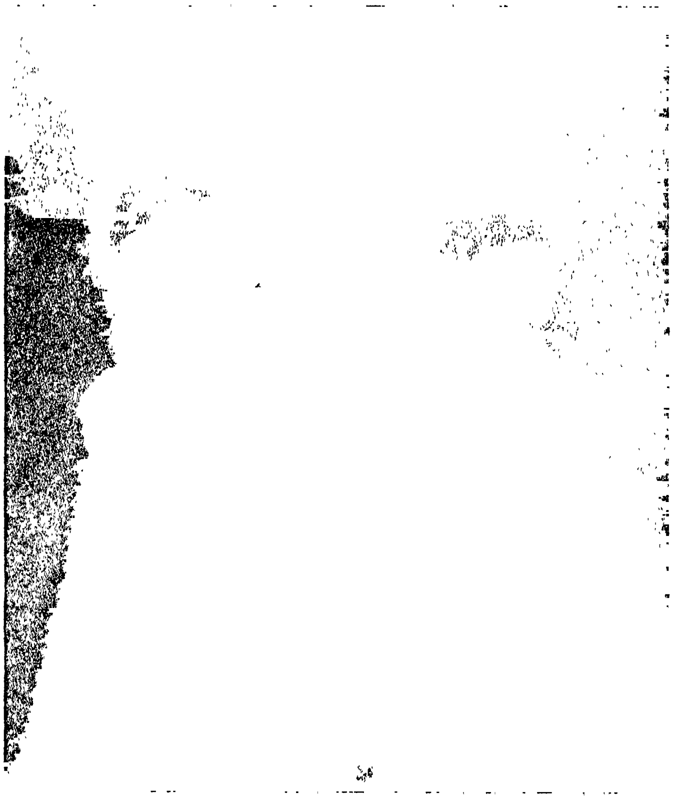


FIG. 15.—MODERATE OBSTRUCTION TERMINAL ILEUM OF APPENDICULAR ORIGIN. OPERATIVE CONFIRMATION.

Accumulations of fecal matter in various parts of the colon also occur as a result of inflammatory conditions outside of the colon, such as chronic appendicitis, gall-bladder infections, etc.

The inflammatory or adhesive processes about the cecum forming after attacks of appendicitis are of great interest in the production of fecal stasis. In the great majority of these cases the appendix has ruptured and the resulting localized peritonitis has bound down the cecum, thereby crippling its motor apparatus. Bands may partially obstruct the ileum. The resulting constipation is exceedingly intractable (Figs. 15, 16 and 17).

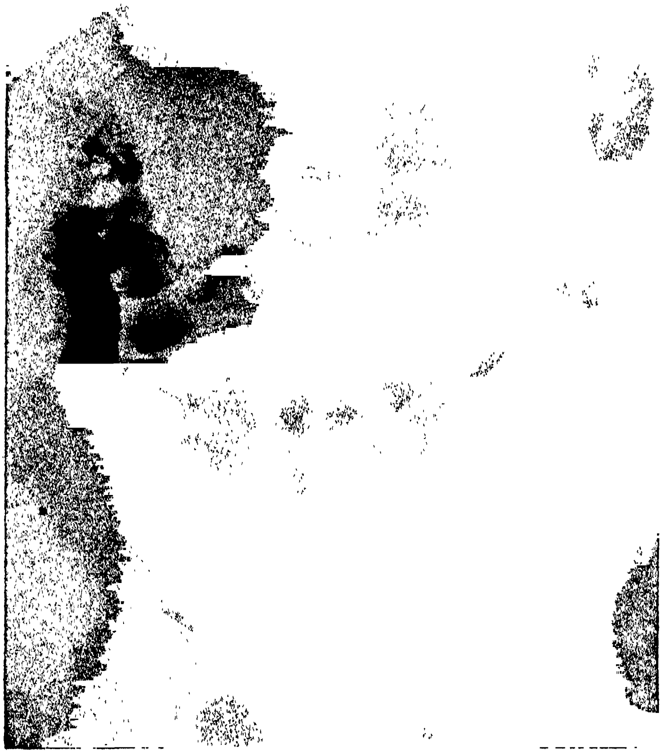
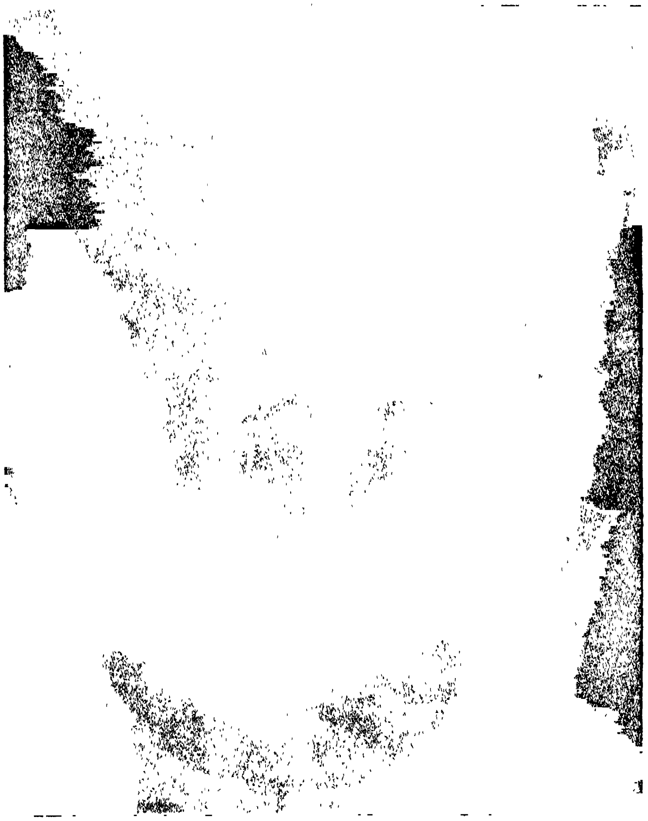


FIG. 16.—HIGH-GRADE OBSTRUCTION WITH SECONDARY DILATATION OF SMALL INTESTINE FOLLOWING ORGANIC OBSTRUCTION. HERE POSTOPERATIVE ADHESIONS ASSOCIATED WITH POSTOPERATIVE HERNIA. HERRINGBONE OR RIBBED PATTERN HERE SHOWN CHARACTERISTIC OF SMALL INTESTINAL OBSTRUCTION. OPERATIVE CONFIRMATION.

In some of these cases appendectomy alone does not suffice to clear up the resultant stasis. Quite frequently we find a stump of the appendix left in the mass of adhesions.

Inflammatory processes resulting from duodenal ulcer and gallbladder perforations may involve the ascending colon and hepatic flexure in such a manner as to cause obstinate constipation. In cases of this character the resultant inflammatory process is not sufficient to cause any obstruction of the bowel by bands or adhesions. The disturbance in motility is largely inhibitory in character. This is particularly true in cases where the stump of an appendix has been left as mentioned (Fig. 18).

Spasmodic contractures involving the iliac colon and rectosigmoid apparatus form temporary obstruction of the colon that may be so strong as to simulate organic disease of the bowel. The resulting constipation is very obstinate. The stools are fragmentary in character, often consisting of small, hard balls. An enema of water produces severe pain with but little effect in securing an evacuation. The patient complains of pain in the lower quadrant. Cathartics accentuate this pain. Upon palpation the iliac colon is felt as a hard unyielding cord which does not relax. Normally the iliac colon alternately relaxes and contracts under the palpating fingers. In atonic conditions of the iliac colon no contracture can be elicited by palpation. When the contrac-



. 17.—Profound Colonic Stasis Associated with Inflammatory Exudate Involving Cecum. Plate at 60 Hours with but One Slight Defecation in That Period. Operative Confirmation.

ture is located at the pelvirectal angle or rectosigmoid apparatus, as Mayo designates it, the diagnosis can be made only by means of the sigmoidoscope. Direct applications of magnesium sulphate solution are of great value in overcoming these contractures. If used systematically definite cure can be effected.

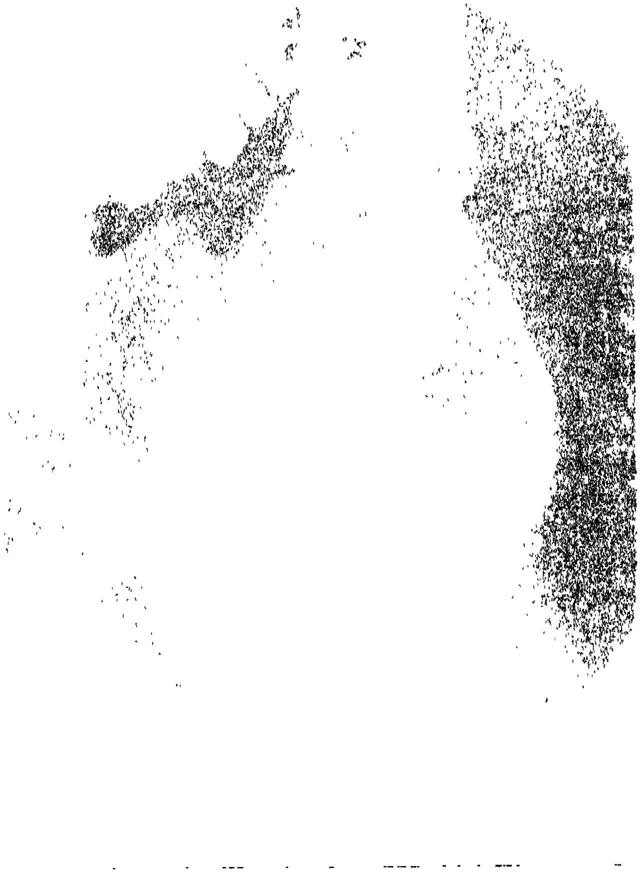


FIG. 18.—COLONIC OBSTRUCTION ASSOCIATED WITH EXUDATE INVOLVING TERMINAL CECUM AND PROXIMAL HALF OF APPENDIX, DISTAL HALF OF APPENDIX REMOVED SOME YEARS PREVIOUSLY, PROXIMAL PORTION BEING MISSED IN MASS OF ADHESIONS. CASE OF SITUS TRANSVERSUS ALSO. OPERATIVE CONFIRMATION.

Atonic conditions of the lower colon, particularly those involving the iliac and pelvic colons, also produce a form of constipation of so severe a character as to produce obstructive symptoms.

CHAPTER XXIII

CONSTIPATION, MUCOUS COLITIS, INTESTINAL TOXEMIA, AND INTESTINAL STASIS

By William Gerry Morgan, M.D.

Constipation, p. 667:—Definition, p. 667; Etiology, p. 668; Symptomatology, p. 671; Treatment, p. 672; Prognosis, p. 677.
Mucous colitis, p. 677:—Treatment, p. 677.
Intestinal toxemia, p. 680.
Intestinal stasis, p. 681.

CONSTIPATION

Definition.—By constipation is understood insufficient fecal evacuation. This insufficiency may relate to the quantity evacuated or to the frequency of evacuation. Modern conditions have established a rule of one bowel evacuation daily, which in a large majority of individuals occurs after the morning meal.

Within normal limits there is a variability in the number of bowel evacuations. A small percentage of people have habitually two or three evacuations daily all their lives, while occasionally an individual is met with who has habitually a bowel evacuation every other day or every third day. In every other respect these individuals show no abnormality; therefore we are forced to look on this variability as physiological. On the other hand, not infrequently the number of evacuations may remain normal but the quantity may be diminished so that the bowels are not completely evacuated and stagnation of feces occurs.

When bowel movements occur less frequently than once in three days, or more frequently than two or three times in one day, the condition must be considered as pathological. The quantity of feces varies to a great extent, depending upon the character of the diet, but in normal individuals it ranges from 100 to 250 grams daily, being greater in amount after a vegetable diet, and less after an animal diet. A large proportion of the dejecta consists of microörganisms, the number being estimated at about 100,000,000 per diem. This accounts for the fact that during starvation, daily evacuations of considerable quantities of feces may take place. The main reservoir of feces is the sigmoid and the upper rectum. In some instances, although a bowel evacuation of normal quantity is expelled daily, yet the duration of time during its progress through the colon has been so prolonged (from one to three days) that the feces is abnormally dry and hard, in which instance the condition may be considered latent constipation. In all individuals once in twenty-four hours feces passes out of the sigmoid and upper rectum down into the ampulla of the rectum which stimulates a desire for defecation. This action is brought about by voluntary relaxation of the sphincter and by the pressure of the abdominal walls during deep inspiration. Normally the act is accompanied by rather a pleasant sensation, never by discomfort.

Etiology.—Habitual constipation is a purely functional disorder in probably 85 per cent. of all cases, and in nearly all of these it is an acquired condition due largely to faulty habits. There can be little doubt but that the tendency of modern life has a powerful influence on increasing the number of those suffering from constipation. The refinement in the character of food, the lessened consumption of drinking water, the advent of motor vehicles, and the tendency to drift to cities where there is lessened bodily exercise, have all contributed to the development of constipation.

Formerly this condition was largely confined to the female, but lately quite as many men as women suffer from torpid bowels. In point of fact, a smaller percentage of women in general at the present time suffer from habitual constipation than was the case ten or twenty years ago. This has come about through reforms in dress and increased outdoor activities, the latter of which induces the drinking of more water and the eating of more food. Formerly pregnancy almost always resulted in chronic constipation, but of late better care during pregnancy and after has largely prevented this condition. Again with more advanced sociological education, women are emancipated from a sense of false modesty, and attend to the calls of nature at the proper time. The repeated failure to obey the desire to defecate by blunting the sensibility of the terminal nerves of the sigmoid and rectum, is a fruitful and a common cause of constipation. Anemia and chlorosis, and the too long-continued use of highly stimulating and irritating substances, such as bran, shredded wheat, figs and the like, are also causative agents in blunting the terminal nerves of the gut. These in addition have a tendency to set up an intractable catarrhal process. Other causes acting in the same way are excessive habitual use of tobacco and alcoholic beverages. The habit of reading at toilet, which at first may seem beneficial, has a tendency to lessen sensitiveness of the rectal nerves, and leads to prolonged sitting at stool, with engorgement of the local venous circulation, and the development of hemorrhoids. Irregularity in the time of going to stool is a frequent cause of beginning chronic constipation.

Perhaps the best special division of constipation is offered by Einhorn, who divides the cases into three groups: (1) Constipation due to a retarded intestinal peristalsis (atony of the bowel); (2) constipation due to a spasmodic contraction of certain portions of the gut (enterospasm, spastic constipation); and (3) constipation depending upon abnormal conditions of other organs.

Until the advent of roentgen rays, Group 1 was considered to comprise by far the majority of the cases. At the present time we know that spastic constipation is much more frequent. Constipation due to atony usually occurs in the young, and begins most often as the result of habitual neglect to respond to the call of nature giving rise to irregular bowel habit, which later gradually drifts into habitual constipation. In atonic constipation, the cecum is the part of the colon most frequently effected, and in the majority of cases it is the seat of more or less pronounced fecal stasis. Less frequently the sigmoid is atonic, dilated and the seat of stasis, and the site of a pronounced catarrhal process, which is difficult to cure. Exhaustion of the general nervous system, through its effect in decreasing intestinal secretion and lessening muscular power, is responsible for many cases of this group. Chronic constipation is found pretty generally distributed in all walks of life, no one class being more subject to it than another, as was formerly the case.

Constipation due to spasmodic contraction of different portions of the intestines was recognized by Einhorn in 1889, long before the advent of the roentgen ray studies of the alimentary tract. This spasmodic contraction affects usually both the circular and longitudinal fibers of the bowel, is of variable duration and at times may almost entirely occlude the lumen of the gut, thus producing temporary partial stenosis.

The exciting causes for this spasm are localized hypersensitive areas in the mucous membrane lining the gut. These hypersensitive areas are irritated by unsuitable food, or are overstimulated by the normal fecal content, thereby causing a spasmodic contraction of the gut at that point. The underlying causes for the development of these hypersensitive areas are those conditions which produce a lowered tone of the nervous system, such as neurasthenia, chlorosis, certain nutritional diseases in those individuals who are overworked and at the same time subjected to severe mental strain, as well as certain disease involving the spinal meninges, the pons and medulla oblongata. A very pronounced type of this condition is seen in persons suffering from chronic lead poisoning.

Spasmodic contraction of the bowels sometimes involves the small gut, but in the case under consideration the colon is chiefly concerned. Any portion of the colon from the sigmoid to the rectum may be the site of this spasmodic contraction, which usually is limited to a small segment of the gut; rarely the entire colon may be in spasm. A not infrequent and usually overlooked cause for constipation is spasm of the sphincter ani. The mucous membrane of the anus is hypersensitive from one cause or another, which prevents voluntary relaxation at the time of defecation, thus retarding the passage of the feces. In the early stages of this type of constipation, the unexpelled fecal matter is returned from the rectum to the sigmoid. This produces stasis and later on dilation of the gut which results in habitual constipation. In these cases a cure is readily effected by proper treatment directed to the anus.

Constipation Depending upon Diseases of Other Organs.—Among the many diseases which are accompanied by more or less chronic constipation are hyperthyroidism and hypopituitarism, and diseases of the lungs, heart, liver, and kidneys. In these diseases constipation is brought about by engorgement of the portal circulation and hyperemia of the intestinal mucosa. Diabetes mellitus is almost invariably accompanied by obstinate constipation, due to the polyuria which drains the system of water and leaves the feces in a dry condition, as well as to the character of the diet which is largely restricted to animal foods. Nearly all of the febrile diseases are accompanied by acute constipation which in a certain percentage of cases leaves the individual in a state of chronic constipation. Diseases in which there is profound disturbance of the central nervous system are usually accompanied by constipation. This is due either to irritation of the nerves controlling the centers of defecation, or to diminished sensibility of the terminal nerves supplying the intestines, so that they fail to respond to the normal stimuli. Chief among these are ulcer, cancer, atony, hyperchlorhydria and catarrhal conditions, as well as ischochymia and a certain definite group of achylia gastrica cases. Constipation in these cases may be due to the character of the chyme which influences the quality and quantity of the various intestinal secretions.

Further causes for constipation are tumors obstructing the lumen, either within or without the bowel, adjacent organs pressing upon the bowels causing partial stenosis of the intestines, and adhesions of the bowel, or of the bowel to the peritoneum. Other frequent causes of constipation are: Visceroptosis with angulation of the gut at one point or another; chronic appendicitis; diseases of the gall-bladder, either through changes in the bile itself or by interference with the flow of bile into the intestine; and catarrh of the small intestines. Although ulcers of the small intestines are associated with constipation, ulcers of the large bowel are usually accompanied by diarrhea, with the exception of dysenteric ulcers, which in the majority of cases produce constipation. Ileal regurgitation is almost invariably accompanied by constipation.

There are two predominant predisposing causes of constipation: First, the character and quantity of food, and the amount of physical exercise; second, repeated neglect of the call of nature, and repeated suppression of the desire for defecation. During the past decade there has been a growing tendency toward refinement of food, which has brought about a lessened fecal residue, thus reducing the size of the bolus with consequent lessening of its stimulating effect upon the bowel. The largely increased consumption of sugar is without doubt a considerable factor in the widespread prevalence of constipation.

The lack of exercise at the present time is not as frequent a cause of constipation as it was in the past. An increasing number of people are seeking outdoor exercise in one form or another and more attention is being paid to affording those living in cities opportunity for such exercise than heretofore. Too prolonged exercise causing fatigue results in constipation; for this reason caution should be used in recommending excessive amounts of exercise to those leading sedentary lives.

The widespread use of cathartics, if continued over a considerable length of time, will produce constipation by rendering the bowel insensitive to the normal stimuli. Often as the result of some acute illness, the bowel becomes temporarily sluggish. If left alone the constipation will spontaneously disappear, and the bowel actions become regular and normal. Yet in eagerness to overcome the condition cathartics are frequently too readily resorted to, with the result that habitual constipation is established.

The fear of intestinal auto-intoxication leads many to turn to the use of purgative drugs, than which at the present time there is no more widespread fetish. Without actual disease of the mucosa, focal retention cannot cause intestinal auto-intoxication, and result in the recognized symptom complex of that disorder. It has been recognized for generations that the healthy colon mucosa is impervious to toxic substances. In our efforts to overcome constipation we are apt to lose sight of this fact, and order for these individuals cathartics, coarse food, and hard medicaments which have the effect of destroying the natural defences to those harmful substances.

Symptomatology.—Chronic constipation in which there is no injury of the mucosa of the colon or rectum does not in the majority of cases produce any subjective symptoms. Since in many instances the mucous membrane is the seat of chronic inflammatory changes, continued constipation produces such subjective symptoms as a feeling of distention and pressure in the abdomen, passing of flatus, borborygmi, and abdominal pyrosis. At times colicky pains of more or less severity are experienced, especially in those persons in whom the feces becomes dry and hard. The abdomen is uniformly distended or the distention may be localized in one portion or another, more frequently in the cecal and sigmoid regions.

Among the gastric symptoms which may be present are loss of appetite, fullness after meals, bad taste in the mouth, coated tongue, belching, pyrosis and mild nausea. In addition to the foregoing there may be a feeling of despondency, mild vertigo, fullness in the head, insomnia, headache, palpitation of the heart with irregularity of the heart action; if the constipation is severe, diarrhea alternating with constipation may eventuate. In rare instances, if the fecal matter is dry and hard and retained for a considerable length of time, severe catarrhal inflammation may be set up and may even result in the formation of ulcers, local peritonitis and perforation of the intestines.

One of the most serious symptoms which may develop in chronic constipation is fecal colic. These cases present intense colicky pains which are due to the effort of the intestines to expel the hardened fecal matter. These pains usually begin at some point along the course of the colon, are more or less intermittent and are often accompanied by localized points of tenderness. The pains usually disappear with the evacuation of the bowels. Passing of flatus gives temporary relief but the pains reappear to permanently subside only after a thorough bowel evacuation. Colicky pains are met with only in case of constipation of the spastic type, and may occur even though incomplete daily bowel evacuations have occurred. In these cases there are almost invariably considerable accumulations of fecal matter in the form of small hard balls similar to goat dung. Often these balls may be felt through the abdominal walls, and even seen in enema solutions which have been passed. The majority of these cases terminate by spontaneous evacuation, the fecal matter being in small hard masses covered with mucus. In a small percentage of cases the constipation results in an acute attack of diarrhea, which is due to irritation of the intestinal mucosa by the hardened fecal matter. In some cases there is no attempt on the part of the gut to spontaneous evacuation, so that some artificial aid is necessary, such as enemas or cathartics.

Constipation may produce a very mild catarrhal process in the bowels, or on the other hand it may result in definite anatomical lesions such as stercoral ulcers, localized peritonitis, or in rare instances, even in perforation of the intestines. In very rare cases in weakened and emaciated individuals, especially in the aged, total paralysis of the intestines may occur with fatal termination.

Fecal tumors are not infrequent in cases of chronic constipation. The cecum, the rectum and the sigmoid flexure of the colon are the most frequent sites for these tumors. Such tumors may vary in size up to several pounds in weight and as a rule are easily recognized. Such large fecal accumulations necessarily produce more or less dilatation of the colon, which at times remains as permanent diverticuli. In these cases the patient frequently has what appears to be normal daily evacuations. The fecal tumors also drag down and cause displacements of the colon.

In some cases of chronic constipation attacks occur in which there is an elevation of temperature accompanied by colicky pains of a more severe type. The fever and pains subside almost immediately after thorough evacuation by means of enemas, leaving behind a localized spot of tenderness which persists for some days. In rare instances there is a moderate leukocytosis which also subsides *pari passu* with the pain and fever. In these cases it is reasonable to suppose that there is a mild localized peritonitis at the point of spasm of the gut.

The stools of those suffering from constipation vary in character and size within wide limits, ranging all the way from the goat dung and pencil stools of spastic constipation to large round masses the size of an orange. In atonic constipation these masses often have to be removed by the finger or with a scoop; at times this causes laceration of the anal tissue.

Treatment.—Persons who have an habitual bowel movement every day or every second day or every third and yet are in perfect health, should be left alone. A one-sided diet should be avoided, inasmuch as exclusion of vegetables, fruits, starchy food and fats tends to produce constipation. Cathartics should not be administered in slight temporary disturbances of digestion, but nature should be permitted to take her course even though the bowels should not move for several days. If left alone, spontaneous bowel evacuation will take place.

Constipation is best avoided by regularity in time of going to stool, by outdoor exercise, by the consumption of a normal amount of drinking water, as well as by regularity in the time of eating and sleeping. **Allaying the patient's anxiety** to have a movement is of the very greatest importance in the cure of constipation. He should be taught not to worry if the bowels fail to act for several days. At the same time he should be instructed that such omission will produce no harmful results. This method of itself will prove effectual in many cases. In the meantime, however, **dietetic** and **hygienic** measures should be instituted and the patient advised to go to stool at a regular time, preferably after the morning meal. He should be told not to strain and to remain at stool not longer than five minutes. If the attempt is unsuccessful, he should wait until the following morning. Often the drinking of 6 or 8 ounces of cold water immediately before going to stool will produce good results.

Diet is of the utmost importance in the treatment of constipation. The diet should consist of those foods which increase peristalsis and stimulate biliary and intestinal secretion, and should exclude substances inhibiting such action. It is important that the patient drink one-half pint of water with meals, which should be sipped between mouthfuls or drunk at the close of the meal.

The diet for spastic constipation should differ from that given in other forms of constipation. Rough and overstimulating foods should be rigidly excluded. These foods include bran, shredded wheat, whole wheat bread, prune spins, raspberries, strawberries, dates, figs, blackberries, nuts, etc., as well as cabbage, spinach, Brussels sprouts, and the like. The diet in these cases should consist of bland bulky foods, such as white breads, rice, grits, farina, macaroni, oatmeal, eggs (always soft cooked), most kinds of fresh fish, all meats except fresh pork, stewed rhubarb, pears, apples, pineapple, bananas, grapes, oranges, Avrocado pears, strained cranberries, prunes put through a sieve, watermelon, jellies, honey in the comb (the comb itself to be eaten), lemonade, orangeade, peaches, fresh asparagus tips, stewed celery, string beans, baked egg plant, carrots, cymlings, fresh tender green peas, squash, beets, stewed cucumbers, small white onions, white and sweet potatoes; among the fats, butter, olive oil, cream, cream cheese and cottage cheese; and simple desserts, preference being given to those sweetened with honey.

In atonic constipation the following foods may be eaten in addition to those suggested for spastic constipation: whole wheat, all cooked cereals, rye and Graham bread, raw chopped cabbage, turnip tops, cauliflower, Brussels sprouts, salads made from tender lettuce, endive or romaine with any of the fruits or vegetables allowed; all kinds of berries except blackberries and blueberries; and a limited amount of cider, buttermilk, sour milk, fermented milk and carbonated waters.

The following should be avoided in all forms of constipation: tea, coffee, cocoa, blackberries, blueberries, old cheese, salt meats, salt fish, spices, condiments of every sort, liquors, red wine and ginger ale.

MECHANOTHERAPY.—In a large proportion of cases of constipation mechanical measures are not necessary. In those of sedentary habits or those who are debilitated or suffer from visceroptosis, some form of mechanical means is often of aid in overcoming constipation. Such measures serve to build up the musculature of the bowel, as well as of the abdominal walls. **Massage** is the method most frequently employed, but since it is contra-indicated in spastic constipation, its use should be confined to the atonic type. Massage should always be administered by one skilled in the method, otherwise harm may result therefrom. The duration of the treatment may be from five to fifteen minutes once daily for several weeks, and gradually discontinued, but never abruptly terminated. Where a skilled masseur is not available, the patient may be taught **automassage** which in some cases may be applied with benefit. The cannon ball method introduced by Sahli may be tried. This consists of rolling a cannon ball, weighing from three to five pounds and covered with flannel, along the course of the colon, from the right iliac fossa to the left, and repeated from thirty to sixty times every morning upon rising.

Gymnastic exercises which develop the muscles of the abdomen are of the greatest benefit. Among these are horseback riding, tennis, golf, basketball, boxing, rowing, fencing and baseball. Straightaway walking is seldom of benefit as is shown by the prevalence of constipation among letter carriers and those of kindred occupations. For the great majority who cannot take one or other of these forms of exercise, some systematic scheme of **home calisthenics** should be arranged. Perhaps the best of those recommended is the set arranged by Illoway and published in his book many years ago. These exercises should be performed on arising in the morning and continued daily for many months. In all forms of exercise, moderation is necessary, and in case of excessive sweating an added amount of water must be drunk to make up for the loss of body fluids.

Electricity is valueless except in atonic constipation, and even in these cases its usefulness is limited. The **faradic current** is most generally employed. For specific direction as to its application, recourse should be had to one or other of the text books on electrotherapy.

Hydrotherapy is sometimes a useful adjunct to other measures, but its successful application is necessarily for the most part confined to hospitals or sanitariums. The **alternating hot and fan douche of Charot,** if correctly applied, relaxes the spasms of the gut in spastic constipation, whereas the **cold jet of Hackel** is recommended as an aid in atony of the bowel. Cold **sitz-baths** as well as the **Priessnitz bandage** are useful in certain cases of the spastic type.

In the treatment of chronic constipation it is not advisable to use injections of irritating solutions, or those which have the effect of producing undue dryness of the mucosa, such as soap, glycerin, vinegar, epsom salts and the like. Such solutions if ever to be used should be reserved for acute constipation. The same is true of the continued use of water alone or water with the addition of salt or soda. The best of all fluids to be used in enemas is the purest quality of **olive oil** which may be injected in rather large quantities, a pint or a pint and a half in the early morning and retained for some hours, or 4 to 8 ounces should be injected at bedtime to be retained all night. On the following morning, as a rule, the oil is passed by itself and followed soon after by the feces. These injections should be given every night or every alternate night until a cure is effected. This form of injections is specially adapted for the relief of spastic constipation.

When the constipation is accompanied by more or less marked catarrhal inflammation, the addition of a small quantity of **ichthyol** in proportion of from one-half to one teaspoonful of pure ichthyol to six ounces of olive oil is beneficial. Another bland and effectual enema is a decoction of **flaxseed.** Two ounces of flaxseed are added to two quarts of cold water and boiled for ten minutes, strained and cooled to body temperature. To this is added one-half teaspoonful of **fluid extract of hydrastis,** and a quart of the solution is injected into the bowel and allowed to remain from fifteen minutes to two hours. The solution which the author has found to be most uniformly useful is **argyrol** in tepid water in proportion of 4 grams (61.73 grains) to 1500 c.c. This solution is especially beneficial in the spastic type. For simple cleansing a solution of tepid water containing **sodium bicarbonate** and **witch hazel** is advised. And still another simple means, one especially adapted to those cases in which there is catarrhal proctitis, is the insertion at bedtime of an **iodoresorcinsulphonate of bismuth suppository,** or any other of similar composition. This is allowed to remain all night; on the following morning another suppository is inserted and allowed to remain until after breakfast at which time a bowel movement follows. Incidentally, this form of treatment has a tendency to relieve engorgement of the hemorrhoidal vessels and also spasm of the sphincter ani. All the foregoing methods may be continued for a considerable length of time if necessary without harmful results. In cases where there is chronic proctitis, injections of one pint of cold water containing 10 or 15 drops of **tincture of iodin** are helpful. The use of boric acid and similar medicaments applied directly to the rectum is not advisable in chronic constipation.

In the majority of cases of chronic constipation purgatives are not required and frequently defeat the ultimate object for which they are given, that is, a permanent cure. In some cases, however, mild laxatives are required temporarily; seldom, if ever, are drastic purges advisable in uncomplicated chronic constipation.

In the spastic form one of the antispasmodic drugs such as **belladonna, valerian, lupulin,** and **sumbul** is useful, especially when combined with the use of one or other of the lenitive enemas.

In the atonic form, in addition to a diet, giving an excess of **ballast agar-agar** or **agar** impregnated with small doses of **phenolphthalein** is useful. More rarely, and for a very limited period, agar combined with **cascara, rhubarb** or **aloes** may be indicated. The author has obtained the best results by giving the agar at bedtime; it is to be taken dry and washed down with cold water. Administered in this way it is somewhat more effectual than when mixed with food or soaked in water, as is more generally advised.

Where the use of cathartic remedies appears to be unavoidable, the mildest forms should be chosen and administered in progressively decreasing doses. Among the many purgatives recommended are aloes, cascara, rhubarb, **compound licorice powder, podophyllin, magnesium**

salts, senna and the **saline mineral waters.** One of the most useful combinations in simple habitual constipation is that suggested by Einhorn:

℞
Pulveris radicis rhei
Magnesiæ ustæ
Sodii bicarbonatis .... āā 20 grams (308.64 grains)
M. Ft. chartæ. D. ad scatulam.
Sig.—One teaspoonful two or three times a day.

The widely used aloes, belladonna and strychnin pill is a very efficient remedy. Another effective combination suggested by Einhorn is:

℞
Podophyllin .... 0.3 gram (4.65 grains)
Extracti physostigmatis
Extracti nucis vomicæ ....āā 0.5 gram (7.716 grains)
M. Fiant cum extracti et pulveris glycyrrhizæ q.s. pilulas No. xxx.
Sig.—One pill twice daily.

Where cascara sagrada is to be used, the fluid extract should be chosen. The most efficient method of administration is to give from 5 to 20 drops in 2 ounces of cold water before meals, three times a day, and gradually reduce each dose. Mineral waters, when resorted to, should be taken in the early morning on an empty stomach and must not be continued for longer than two or three consecutive weeks. A useful pill suggested by Kemp for cases in which constipation is accompanied by more or less anemia is:

℞
Blaud's pill .... 0.324 gram (5 grains)
Aloini .... 0.032 gram (½ grain)
M. Ft. pillula No. 1.
Sig.—One pill after meals three times daily.

Constipation of pregnancy should be relieved by mild cathartics rather than by the use of enemas. Perhaps the most useful are cascara sagrada, and compound licorice powder administered at night. In these cases care should be exercised not to produce liquid stools. In constipation due to spasm of the sphincter ani, gradual or rapid dilation may be resorted to; this is often all that is required to effect a cure of the constipation.

Acute constipation may be due to complete obstruction of the intestinal tract, to paresis of the intestine, or it may accompany one or the other of the acute febrile diseases. Perhaps the most common cause is acute appendicitis. In these cases an accurate diagnosis is necessary with active measures for relief of the underlying disease.

In fecal tumors or fecal impaction purgatives are contra-indicated until a large part of the fecal accumulation has been softened or has been removed by copious injections of solutions of soda or of olive oil and **glycerin,** or water with glycerin. At times it may be necessary to resort to solutions of water containing **ox gall** and water. In impactions in the rectum, it is sometimes necessary to break up the fecal masses with the finger or some instrument such as the scoop; in doing this great care should be used not to injure the bowel. After the fecal masses have been reduced to a size capable of being expelled, doses of **castor oil**, or castor oil and glycerin in combination with suitable doses of **laudanum** and **belladonna** may be administered.

In fecal fever a correct diagnosis as to the cause of the condition is imperative. As a rule it is associated with spastic constipation and the treatment advised for the latter will relieve the fever and the localized peritonitis which usually accompanies it. If stercoral ulcers are present, they will require appropriate treatment for their cure.

Prognosis.—A favorable outcome may be looked for in the majority of cases of constipation, regardless of age and duration of the disease.

MUCOUS COLITIS

Mucous colitis is a disease of the intestine in which mucus is passed either alone or with the feces in jelly-like masses or in the form of tough pseudo-membrane resembling tapeworm, and sometimes as a complete cast of the bowel. These membranes and casts consist of true mucus, fibrin never being found in them.

The subjective symptoms accompanying mucous colitis range all the way from the slight, more or less transient distress and tenderness along the course of the colon, with slight mucous discharges on the one hand, to the severe attacks with fever and abdominal pains and distress, and with copious mucous discharges from the bowels. The symptom complex may be so misleading as to confuse the diagnosis unless the stools are carefully examined. When this is done, it becomes simple.

There are two forms of mucous colitis, one in which there is pain along the course of the colon with tendency to diarrhea, and the second in which the patient is constipated and has more or less copious discharges of mucus and severe pain amounting at times to colic. This class always occurs in nervous individuals. Clinically it is not always possible to make this sharp distinction, since not infrequently in mucous colitis there is constipation alternating with diarrhea which does not readily yield to treatment.

Treatment.—To be successful treatment of mucous colitis must have a twofold object: first, to heal the bowel; and second, to relieve the underlying nervous disorder.

The first or local treatment must protect the mucosa from further irritation and promote healing of the inflamed membrane. The first object is attained through a carefully arranged **diet**, and by the use of **colonic irrigations**, and **enemas** of **pure olive oil** from which the fatty acids have been removed. The saline mineral waters, castor oil, and other drastic purges are rarely, if ever, called for, and usually increase the patient's suffering. When laxatives are necessary, only the milder ones should be selected, and they should always be combined with efficient antispasmodics such as **belladonna**, **sumbul**, or **valerian** and should not be continued longer than is imperative.

The abdominal pain and distress in the majority of patients can be relieved by **rest in bed, hot fomentations** to the abdomen or the application of the modified **Priessnitz bandage.** In more severe attacks the addition of a suppository containing **veronal,** 5 grains (0.324 gram), with aqueous extract of belladonna, 1/6 grain (0.0108 gram); one suppository in twelve hours will usually allay the pain and render the use of opium or its derivatives unnecessary, and does not have a constipating effect.

Irritating residue must be rigorously excluded from the diet of these patients. This does not preclude anticonstipating diets. The so-called b.b. dietary of our local hospitals consists of very bland but bulky foods with ample ballast, but which does not irritate the mucosa, and is sufficient to stimulate the muscular fibers of the gut. The following is suggested as a type of a diet suitable to uncomplicated cases of mucous colitis:

7 A. M. (in bed), 8 ounces of cold water to which has been added 1/8 of a teaspoonful of table salt; or 6 ounces of *Vichy Celestines* taken at body temperature.

7:15 A. M., abdominal gymnastics followed by a cold towel bath. This bath consists of rubbing the body briskly with a rough Turkish towel wrung out of cold water, then drying with a rough dry towel. This bath should be taken with the patient in standing warm water.

8:00 A. M., breakfast, juice of an orange or of one-half of a grapefruit without sugar; a dish of farina or cream of wheat both to be boiled four hours and eaten with cream but no sugar; one or two eggs soft cooked; breakfast bacon broiled but not hard or crisp; cold white bread or rolls or muffins, made from white flour or from white flour with Graham flour which has been sifted to remove the bran; a generous amount of butter and bee's honey in the comb, the comb to be eaten as well; and a glass of water.

1 P. M., beef, mutton, lamb, chicken, turkey, squab, quail, capon, guinea, sweetbreads, and ham (meats may be cooked any way except fried, but they must be tender); white potatoes baked or mashed, or macaroni thoroughly cooked; stewed celery, tips of fresh asparagus, string beans, fresh green peas, baked egg plant, stewed carrots, squash, cymlings, fresh cucumbers boiled, boiled young onions, French artichokes, breads with butter, baked or stewed apples or baked pears, or baked or stewed peaches, baked bananas, Avocado pears, grapes if the skins and seeds are carefully rejected, grapefruit and oranges.

6:30 P. M., some good soup without grease or spices or condiments; otherwise the meal should consist of foods suggested for midday meal; fresh fish may be substituted for meat once a day if desired.

The foregoing diet is suitable for the most severe cases of mucous colitis. The author does not agree with those writers who include from the start moderate irritating foods, and advise progressively increasing them in the course of treatment. He is more inclined to increase the amounts of the bulky but bland elements of the dietary.

Colonic irrigations, if properly carried out, are indispensable in the treatment of mucous colitis. The washing out of the excess of mucus and decomposed intestinal detritus is of importance, principally in order that the healing medicaments contained in the solution may come into more intimate contact with the mucosa. In the course of more than ten years' practice the solution which has proven of the greatest value to the author is varying strength of **argyrol** in water at a temperature of 94° F. (34.44°C.). These enemas are given preferably in the evening or at bedtime in order that the patient may have a longer rest thereafter. The

only correct method of administering colonic irrigations is as follows: The patient is placed upon his back with the hips slightly elevated and the solution is allowed to run into the bowel slowly from a glass or enamel irrigative receptacle placed about 18 inches above the level of the body. When two-thirds of the solution has passed in, gentle succussion palpation is practiced along the course of the colon from the left iliac region, which facilitates the onward flow of the solution and brings it into more intimate contact with the mucous membrane. The solution should be retained from five to fifteen minutes according to the hypersensibility of the bowel. The soft rubber rectal and colon tubes are no longer used, glass irrigating points 4 inches long being all that is required. This treatment should be persisted in daily for from ten days to five weeks. In a certain percentage of cases, some time during the course of treatment, probably as soon as the tendency to spastic contraction of the colon is overcome, morning daily stools will occur independent of the irrigation. When this occurs, the irrigations are gradually discontinued. If constipation persists after the disappearance of the excess of mucus from the stools and the tendency to spastic contraction of the colon ceases, recourse may be had to enemas of **olive oil** or to **agar** administered by mouth or it may be necessary to employ both these measures. Olive oil enemas of from 4 to 8 ounces are given at bedtime and are to be retained all night. Where difficulty is experienced in retaining the oil a hypnotic to induce sleep may be given. The oil is expelled in the early morning and is followed later by a stool. The best method for the administration of agar has been given in the section on Constipation.

When mucous colitis is not accomplished by spastic contraction of the colon, the mucous membrane may be healed and the constipation relieved by the addition of **ichthyol** to the olive oil, in proportion of 30 to 60 drops to 6 ounces of olive oil.

Solutions of soda, salt and boric acid are inadvisable as they have a tendency to produce irritation and undue dryness of the mucous membrane. The application of electricity either cutaneously or by enema has not proven of sufficient benefit to warrant the continuance of its use.

The treatment of these cases by the continued administration of salines and castor oil by mouth, and the use of intestinal antiseptics has not proven successful in the author's experience.

The field of usefulness of drug administration by mouth is limited to those usually employed for the relief of the anemia which usually accompanies mucous colitis. In these cases, the administration of **iron** and **arsenic** is necessary. The more modern and perhaps the best method of administration is by daily hypodermic injections.

The neurasthenic symptoms which often occur in mucous colitis require treatment appropriate for this condition.

A permanent cure may be hoped for only where a normal action of the bowels is established and maintained throughout life. As pointed out by Musser: "A bowel movement cannot be considered normal if laxatives of any sort, massage, injections, suppositories, or any other artificial means are used to promote bowel evacuation."

At the present time it is rarely necessary to resort to surgical intervention for the cure of mucous colitis. Formerly there were many writers who advocated appendicostomy as an avenue for irrigation of the colon from above. For a time this had considerable vogue for the treatment of amebic dysentery, as well as for mucous colitis. A later procedure which fortunately was followed but for a brief period was colotomy and cecotomy by which an attempt was made to put the colon at complete rest. The author sees no rational basis for either of these surgical measures and never resorts to them.

INTESTINAL TOXEMIA

Auto-intoxication or true intestinal toxemia, if there is such a disease, is of very rare occurrence. On the other hand, the symptom complex—headaches, sleepiness, weakness, mental hebetude, early fatigue, nervousness, mental depression, insomnia, loss of appetite, etc.—occur constantly in the daily routine of every busy practitioner.

The author believes that Alvarez is correct in his assumption that these symptoms are not toxic, but are pressure symptoms due to distention of the colon with irritation of the mucosa by the fecal accumulation. An additional fact which must be recognized is the individual's mental attitude to constipation. For generations people have been taught to regard constipation as the underlying cause for most of the ills of the body and so exaggerate the symptoms which they have. No convincing proof has been offered to show the relation between the symptom complex of auto-intoxication and the absorption of toxic substances from the bowels.

Many writers on auto-intoxication base their belief on the fact that these symptoms are relieved instantly when the bowels are evacuated. It is difficult to understand how such prompt relief could be obtained by such means in systemic conditions, and the author wishes to go so far as to say that it is impossible. That such relief does take place after a bowel evacuation in the majority of individuals suffering from this symptom complex is without question. This relief can be explained only on Alvarez' assumption.

Absorption of toxic substances from the small gut seldom if ever occurs, as stasis in that part of the intestinal tract is rare and is of short duration. Inasmuch as it is well known that absorption from the bowel can take place only in the presence of liquid contents, and since the feces begins to harden in the ascending colon, the only place where absorption might take place would be in the cecum. It is a well-recognized fact that the cecum is very rarely the seat of chronic inflammation, as occurs so frequently in the distal portions of the colon; hence, one of the necessary factors, namely, a diseased mucosa, is absent.

The treatment for this class of cases is the same as the treatment for mucous colitis or chronic constipation, being followed according to the predominating symptoms as outlined in previous sections.

INTESTINAL STASIS

Intestinal stasis is an abnormal accumulation of intestinal contents in some portion of the gut. This condition is brought about through delayed progress, and is due to inhibition of the nerves supplying both the longitudinal and circular fibers, resulting in the slowing of the intestinal stream, and in a weakening of the musculature with more or less dilation at that point.

The diagnosis is made chiefly by the roentgen ray studies.

Stasis rarely occurs in the duodenum, more commonly in the cecum and sigmoid, and occasionally in the ptosed transverse colon.

Intestinal stasis must be considered as a symptom rather than as a disease entity. It may be due to or an accompaniment of any of the many pathological conditions which interfere with the enervation of the gut, or it may be due to delay in the transmission of the intestinal contents onward. The most common causes are adhesions about the duodenum, chronic appendix with adhesions, dilated or mobile cecum, enteroptosis, adhesions about the gall-bladder, chronic constipation and general debility affecting the nervous system.

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CHAPTER XXIV

DIARRHEA

By Julius Friedenwald, M.D.,
and
Theodore H. Morrison, M.D.

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Definition.—The term diarrhea implies a lessened consistency, and usually an increased frequency of the stools. While the increased frequency need not necessarily indicate the presence of diarrhea alone (as is observed, for example, in the condition known as fragmentary constipation in which numerous solid stools may be present during the day), yet this affection always implies a lessened consistency. On the other hand this decrease in consistency need not be extremely marked, for, even though but a few soft stools containing mucus, pus, or blood are noted in a day, the term diarrhea may be appropriately applied. As a rule there is a combination of both conditions at hand.

Etiology.—Since the term diarrhea is applied only as a symptom denoting the passage of liquid stools, it not only embodies those forms due to disease of the gastro-intestinal tract, but it is also indicative of those varieties which are due to conditions arising outside of this tract, such as one frequently observes in toxic states as well as in functional disturbances of the nervous system.

Usually diarrheic movements are caused by stimulation of the bowels due to increased peristalsis which may be encountered both in the small and large intestine. This may occur even though there be no pathological change in the wall of the bowel itself.

Stimulation of the bowel by irritating substances may cause the fluid contents to be rapidly carried into the colon, preventing absorption in the small intestine or causing increased transudation of the water from the blood into the bowel with increased production of fluid or mucus. Stimulation with peristalsis may also be induced by influences affecting the central nervous system, such as emotion or increased excitability of the nerves of the intestine itself. On the other hand, increase in peristaltic movements with the production of diarrhea may be caused by lesions in the bowel itself, such as inflammatory changes, ulcerations, growths or obstructions. Not infrequently a number of these factors acting together are responsible for the increased peristalsis.

While the factors noted are frequently the cause of a diarrhea, diarrhea itself when prolonged and intense may lead to the production of serious complications. Through the lowering of the general resistance of the body, secondary infections of a more or less severe type may take place, producing such grave complications as septic pneumonia, peritonitis, or ulceration of the bowel with adhesions.

In studying the etiology of diarrhea it is important to determine the intestinal site of this disturbance, and to note whether the intestinal peristalsis of the small intestine is increased with that of the large bowel.

In diarrhea originating in the small intestine there is an acid reaction of the stools, which contain many poorly digested food particles; the mucus is intimately mixed with the feces, and unreduced bile pigment is noted. In diarrhea originating in the large intestine, the stools are usually alkaline in reaction and are well digested; the mucus is free and the bile is reduced.

Diarrheas may be divided into acute and chronic forms.

ACUTE DIARRHEA

Etiology.—Acute diarrhea is usually secondary to a gastro-intestinal catarrh which may be induced by improper food, intoxications and infectious diseases. Nervous influences may cause frequent bowel evacuations without inducing catarrhal conditions, the diarrhea resulting from central or reflex stimulations.

Diarrhea Due to Improper Food.—Indiscretions in diet are very often followed by diarrhea which is the result of an acute gastro-enteritis. The food may be coarse, improperly prepared, insufficiently masticated, taken in too large quantities or ingested either too hot or too cold. Food poisoning from tainted meat, sausage, milk or fish, spoiled vegetables and food infected with various microörganisms are usually accompanied by severe and violent diarrhea. Foods of this character contain at times toxic materials known as ptomains.

DIARRHEA FROM INTOXICATION OTHER THAN FOODS.—This condition may be produced by poisons or medicaments. Some of the drugs that produce intestinal irritation when taken over a long period of time are: Mercury and arsenical preparations, even when given hypodermically; and drastic purges, such as jalap, senna, podophyllin, etc. Poisons such as phosphorus, mineral acids, alkalies and corrosive sublimate may also cause acute diarrhea when taken internally.

DIARRHEA IN ACUTE INFECTIOUS DISEASES.—Acute infectious diseases such as typhoid, measles, cholera, influenza and pneumonia are frequently associated with diarrhea. In these cases it is secondary to the general infection present, and the resultant gastro-enteritis. The specific infections such as dysentery, cholera, and typhoid need not be considered here as these are fully discussed elsewhere.

DIARRHEA FROM NERVOUS INFLUENCES.—This type of diarrhea originates either from excessive stimulation of the nerves governing peristalsis (the motor function) or from the transudation of serous material into the intestinal canal (secretory function), produced by nervous influences. In some cases both factors may be concerned. Numerous watery actions may occur following some emotion such as fright or shock.

Symptomatology.—The symptoms depend largely upon the etiological factors and the intensity of the catarrhal condition produced. The stools are frequent and watery, contain mucus and undigested food; there is diffuse abdominal pain, lack of appetite, malaise and weakness. If there is an associated acute gastritis, nausea and vomiting may overshadow the intestinal condition. In the severe cases, albuminuria and fever are present, and the profuse diarrhea may induce an alarming state of weakness in the young as well as in those advanced in years.

The usual feature of a nervous diarrhea is its sudden onset and equally characteristic is its usually rapid termination. This condition may occur in the nervous as well as in individuals in normal health when overworked or under a strain as, for instance, in students preparing for examinations. The stools in this condition vary in appearance and number. There may be few or many watery thin actions a day with a small amount of mucus and perhaps some undigested food particles.

Treatment.—The principle of treating this condition lies in the removal of the offending material and resting the affected organ, thereby restoring normal function. An initial purgative is usually indicated and in the absence of nausea, **castor oil** or a **saline laxative** is most efficacious. In the presence of an associated acute gastritis in which nausea is a prominent symptom, it is best to give **calomel**, in 1/4 grain (.0162 gram) doses, with *soda* at half-hour intervals for six or eight doses, to be followed later by a saline purge.

Pain may be controlled by hot applications such as **stupes** of various forms and **hot water bags.** Astringents and opiates are interdicted until the intestinal canal has been thoroughly evacuated. **Rest in bed** and

abstinence from food for twelve to twenty-four hours is helpful. The diet should consist of warm broth, cocoa, tea, well-cooked cereals, such as rice, cream of wheat and oatmeal. Milk is not well borne in these cases, at least not in the first few days. Nausea may be allayed by **cracked ice, creosote, lime water**, etc. **Paregoric** in dram doses (3.9 grains), **bismuth subcarbonate** or **subnitrate**, in 10 to 30 grain doses (.65 to 1.95 gram), alone or in combination, can be given every two or three hours until the diarrhea is checked. Should constipation ensue, enemata will be better than purgatives, although mineral oil can be utilized with safety. The diet can be gradually increased after the first day or two, with the addition of toast, gruel, soups, soft eggs, chicken, and baked potato; this will, of course, depend upon the progress of the patient. The diet must be carefully observed for a week or two after the acute attacks, as relapses due to dietary indiscretions are apt to occur. By carelessness in the management this condition may be converted into chronic enterocolitis.

In cases of acute diarrhea in patients of advanced years it is well to use as stimulants small doses of **whisky** or **brandy** along with hot applications; paregoric should also be given in small doses when necessary, and enemata used in preference to purgatives.

In the severe forms of gastro-enteritis, known as cholera morbus, supporting measures such as hot applications to the abdomen and extremities, stimulants in the form of **alcohol** and the hypodermic administration of **morphin** are especially indicated. **Starch and opium enemata**, containing from 25 to 30 drops of **laudanum**, may be used to check the diarrhea if the vomiting persists. The treatment as outlined above may be followed after the vomiting and purging cease.

CHRONIC DIARRHEA

Etiology.—It is rather difficult to arrange a satisfactory classification of the different varieties of chronic diarrhea. This is in part due to the fact that with improved methods of investigation the etiology of hitherto undetermined forms is gradually being made clear, necessitating frequent changes in classification. The difficulty is further increased in that there are still certain varieties the etiology of which has not yet been satisfactorily explained.

A great number of etiological factors have been noted in the production of chronic diarrhea and at times several causes may combine in an individual instance; thus the diarrhea accompanying achylia gastrica is not uncommonly associated with some forms of chronic colitis. Arranged according to the etiology, chronic diarrhea may be due to disturbances of the stomach, pancreas, liver and intestine, or to affections of various organs outside of the gastro-intestinal tract.

The authors have drawn up the following classification of chronic diarrhea based as far as possible on the etiology of this affection:

(1) Disturbance of gastric function—achylia gastrica.
(2) Disturbance of the pancreatic functions.

- (a) Deficiency or absence of pancreatic ferments.
- (b) Cancer of the pancreas.
- (c) Cysts of the pancreas.

(3) Disturbed liver function.
(4) Chronic enterocolitis.
(5) Ulcerative colitis, sigmoiditis and proctitis.
  (a) Entamebic.
  (b) Bacillary.
  (c) Tuberculous.
  (d) Syphilitic.
  (e) Carcinomatous.
  (f) Follicular.
(6) Mucous colitis.
(7) Simple colonic infections.
(8) Intestinal obstruction and stasis.
(9) Chronic appendicitis.
(10) Diverticulitis, involving the sigmoid and rectum, polyposis of the colon.
(11) Lesions of the brain and spinal cord.
(12) Disturbance of the glands of internal secretion.
  (a) Pancreas.
  (b) Suprarenal.
  (c) Thyroid.
(13) Cardiorenal disturbances.
(14) Diseases associated with disturbed metabolism.
(15) Following operative procedures on the gastro-intestinal tract.
  (a) Cholecystectomy.
  (b) Gastro-enterostomy.
  (c) Pyloroplasty.
  (d) Appendectomy.
  (e) Resection of bowel.
(16) Nervous influences, such as anxiety and excitement.

Diarrhea Due to Achylia Gastrica.—Chronic diarrhea is not infrequently due to achylia gastrica, though this condition may even occur as a result of a mere diminution of the gastric secretion, such as one frequently observes in cases of chronic gastritis. On the other hand it is important to note that only about 30 per cent. of patients affected with achylia present diarrhea, a large proportion being associated with constipation. The factors leading to the production of diarrhea in these cases have been variously explained. It has been maintained that due to the lack of digestion in the stomach caused by the absence of hydrochloric acid and diminished pepsin secretion, undigested food is quickly thrown into the bowel and by acting as a mechanical irritant may lead to the production of frequent and liquid stools.

By means of the Schmidt diet test it has been definitely ascertained that the appearance of undigested connective tissue fibers in the stools is indicative of poor gastric digestion. This test is ordinarily positive in gastrogenous diarrhea, indicating that the frequent passage of large quantities of connective tissue, especially following the ingestion of raw meat, is sufficient to stimulate the intestinal mucosa to such a degree as to produce liquid stools. In addition the coarse connective tissue remains permit fermentative and putrefactive changes, caused by the growth of various forms of microörganisms and thus lead to infections and inflammation in the intestines with an increased frequency in the evacuations.

Finally on account of the absence of hydrochloric acid (and this acid is known to stimulate pancreatic activity) the activity of the pancreatic secretion is impaired; this may also result in the production of intestinal fermentation accompanied by diarrhea.

Symptomatology.—In the early period of this affection the symptoms are usually very indefinite, there being but occasional mild attacks of diarrhea; however, in some forms the onset is extremely acute, accompanied frequently with signs of infection. In many instances there is an intermittence in the occurrence of the diarrhea with periods of well-being often extending over days or weeks. The diarrhea, however, soon becomes more frequent until it may be almost constant. In some instances it is, as Vanderhoof has pointed out, of the morning type, most of the stools occurring before noon. Finally, the intestinal mucosa is involved, leading to inflammation of both the small and large bowel. During this entire period gastric symptoms may be absent or so insignificant that they may be entirely overlooked. Usually there is some discomfort in the abdomen which is frequently found distended; relief is usually afforded by bowel evacuations.

The number of stools varies from a few to many a day. They are yellow in appearance, foul smelling, and contain undigested food remains, mucus and even blood in protracted cases. The Schmidt diet test reveals undigested connective tissue.

Diagnosis.—This is finally confirmed by means of a fractional gastric analysis which usually reveals an entire absence of free hydrochloric acid, during the entire period of digestion, *i.e.*, a true achylia.

Treatment.—The treatment of the diarrhea due to achylia gastrica is mainly **dietetic**. Inasmuch as there is an absence of gastric secretion the food should be administered in a thoroughly comminuted form, making as little demand as possible upon the gastric digestion.

Although it is necessary to greatly restrict the diet, sufficient nourishment must be taken, inasmuch as many patients with this affection lose flesh rapidly and are much undernourished. Since raw connective tissue can be digested only by the gastric secretion and not by the intestinal juices, raw and smoked meat must be interdicted. The blood should be given largely in the liquid and semisolid form. Of the liquids, **broths** are especially beneficial. **Vegetables** are best taken in the purée form after removal of the cellulose. Beans and peas are to be recommended when mashed and strained, because they contain considerable quantities of protein. Potatoes and rice should be taken only when well cooked and should be given with milk or broth; stale wheat bread and toast can be eaten with impunity. Eggs are best taken soft boiled, while meats are usually badly borne and should be allowed only in small quantities in the most easily digestible forms; boiled brains, sweetbreads and minced chicken are best. Raw oysters and white fish are allowable. Milk is quite apt to increase the fermentation in the bowel as well as aggravate the diarrhea, and is not to be recommended; malted milk and cocoa can be taken as substitutes.

Rest and **diet** play the most important rôle in the treatment of this affection, while drugs are only of secondary importance. According to the authors' experience, however, **dilute muriatic acid,** when prescribed in 15-drop doses, three or four times at intervals of fifteen minutes after meals, is of great value in the relief of this form of diarrhea. The immediate relief from this treatment is often most striking. Astringents, such as the preparations of bismuth and tannic acid, are useful only as a means of temporary relief.

Diarrhea Due to Disturbances of the Pancreatic Function.—Usually in disturbances of the pancreatic function constipation is noted as a prominent symptom; at times, however, diarrhea is observed. The diarrhea present in achylia gastrica has been attributed partly to a coëxisting deficiency in the pancreatic secretion, but no definite proof of this statement is as yet at hand. In affections of the pancreas there are frequently large irregular stools with alternating constipation and diarrhea. On examination of the stools, after a Schmidt test diet, a diminution or absence of the ferments, trypsin, steapsin and diastase is noted together with the presence of much undigested food.

The presence of the cellular nuclei in the meat remnants which are digested according to Schmidt only by the pancreatic secretion and not by the gastric juice, is additional evidence of disturbed pancreatic function. The deficient steapsin digestion gives rise to large amounts of visible fat in the stools whereas the disturbed amylopsin secretion is noted by the presence of undigested starch remains. A deficiency or absence of the pancreatic ferments is easily determined by means of the method described by Einhorn with the duodenal tube, and which has furnished most satisfactory results in the authors' hands. The presence of pancreatic ferments in normal quantities is evidence pointing against disturbed pancreatic function; a constant absence of one of the ferments is according to Einhorn indicative of chronic pancreatitis.

In addition to the characteristic diarrhea the presence of marked emaciation, colicky pains, and sugar in the urine further denotes pancreatic involvement. Prolonged pancreatic disturbance may produce a secondary catarrh of the bowel, and therefore induce diarrhea.

In carcinoma of the pancreas there is frequently a special train of symptoms pointing directly to this affection. In the earlier stages of this disease, the symptoms referable to disorder of the gastro-intestinal tract are manifested. These are especially loss of appetite, nausea, and vomiting. There is usually constipation, which is rapidly transformed into diarrhea. The diarrhea becomes more and more intense, and occasionally fatty stools are observed. After a longer or shorter period of time jaundice appears, which gradually becomes more and more pronounced. The urine at times contains sugar; the liver enlarges and becomes nodulated, and further evidences of malignancy are noted by the cachexia. This affection is of such frequent occurrence that whenever a persistent diarrhea is observed in persons over forty years of age, which cannot be accounted for by the usual causes, carcinoma of the pancreas should be borne in mind.

In cancer of the pancreas, there is usually a diminution of or an absence of the pancreatic ferments in the duodenal contents, and of the diastatic ferment in the stools, though at times the presence of all of the ferments have been noted. Pancreatic cysts give rise at times to diarrhea not only on account of the disease present in the pancreas, but due to the pressure on the abdominal organs as well.

TREATMENT.—The treatment of diarrhea due to disturbed pancreatic function should whenever possible be directed toward the cause. Much, however, can be accomplished by the **regulation of the diet.** The fats should be greatly restricted. Butter, cream, and oil should be allowed only sparingly. Proteins should also be restricted, the meats allowed only when scraped or minced, and milk and eggs consumed only in moderate amounts. Carbohydrates in the form of cereals, stale wheat bread, mashed or baked potatoes, and vegetables in the purée form are permissible in liberal amounts. For the treatment of the diarrhea **muriatic acid** administered in fairly large quantities with **pepsin** before and after meals is helpful and pancreatic preparations serve an excellent purpose in the relief of this condition.

Diarrhea in Disturbed Liver Function.—In disturbances of the hepatic functions constipation is usually noted, though in a small group of cases diarrhea is observed. In cases described as biliousness or "torpid liver," attention to which has again been more recently attracted because of the studies made possible by the Lyon method of nonsurgical biliary drainage, biliary stasis has been observed in many instances due to infection in atonic gall-bladders. These patients are sallow in appearance, weak, affected with indigestion and usually with constipation and are frequently the subjects of sick headaches and migraine attacks. In a certain group of these cases apparently with the identical symptoms the constipation is replaced by diarrhea.

Again in that form of infective jaundice known as Weil's disease or "bilious typhoid," together with the symptoms of chills, fever, headaches, muscular pain, jaundice, and gastro-intestinal disturbance, diarrhea is usually present.

While constipation is usually observed in active and passive congestion of the liver, attacks of diarrhea are not uncommon. Diarrhea is likewise not unusual in atrophic and hypertrophic cirrhosis of the liver.

TREATMENT.—The treatment of the diarrhea due to disturbed hepatic function must as far as possible be directed to the cause. A **restricted diet** should be insisted on, and **calomel** administered even though diarrhea be present; after this a simple but carefully regulated diet of protein fats and carbohydrates may be allowed. In these affections of the liver in which the liver function is markedly impaired, the fats and carbohydrates must be greatly restricted. In many liver affections overeating is apt to produce diarrhea and the use of alcohol may cause serious manifestations; the use of alcoholic stimulants should, therefore, be avoided. The foods producing an irritating effect on the liver are spices, pickles, peppers, mustard, meat broths, and concentrated meat extracts, onions, radishes, and horseradish; all of these should be forbidden. In serious liver disturbances the diet should usually be limited to broth, diluted or peptonized milk and buttermilk.

In the form of diarrhea which is due to biliary stasis, it is not unusual to observe by means of carefully conducted biliary drainages according to the Lyon method a rapid change in the appearance and strength of the patient, and an improvement in the digestion and diarrhea with complete relief in many instances.

Diarrhea Due to Chronic Intestinal Catarrh.—Chronic enterocolitis frequently results from an untreated or unhealed acute catarrh. It may also be caused by the abuse of laxatives, errors in diet, or it may result from various chronic disturbances of the intestine itself as ulcer, tumor. etc. In this affection there is usually a persistent diarrhea extending over several years with frequent acute attacks associated with pain and tenderness over the colon. The stools are usually thin and watery, offensive and contain considerable mucus, together with blood and pus at times. The number of stools vary from 3 or 4 to 8 or 10 a day, though there may be periods of relief when the stools may become solid and the patient even be constipated. The presence of mucus alone in the stools is not sufficient to warrant the diagnosis of catarrhal enterocolitis without the presence of the other symptoms already mentioned.

TREATMENT.—The treatment of catarrhal enterocolitis is usually satisfactory. **Diet** and **rest** play an important rôle in this direction. The patient should be at rest in bed for a period of several weeks. The diet should be both antiputrefactive as well as antifermentative. It should be restricted as far as possible to broth, cocoa, eggs, toast with butter, minced meats, fish, cereals and puddings. In many instances the administration of **calcium preparations**, together with **bismuth** or some form of **tannic acid** such as **tannigen** is of great help in relieving this condition. In addition most of these patients do well when treated with irrgations; of these **solutions of permanganate of potash, boric acid,** and **gelatin,** and **instillations of protargol** are most valuable.

Intestinal Fermentative Dyspepsia.—In this condition the carbohydrates are digested imperfectly or not at all. This undigested matter undergoes fermentation, becoming acid, and produces gas and gives to the stools, which are diarrheic, a putty, foamy like appearance with very sour odor. This affection is due, according to Schmidt, to a disability of the secretion of the small intestine to digest starches. Symptoms of abdominal pain, gurgling, flatulency, and distention are often noted, In the putrefactive type diarrhea is produced by special articles of food, such as meat, eggs, vegetables and fruit.

In this condition the stools are liquid and putrid in odor and contain undigested food remains. Intestinal fermentative dyspepsia may be a primary disease or it may be secondary to catarrhal affections of the intestine or to achylia gastrica.

In the treatment of this disorder the primary affection requires careful attention. The treatment is largely **dietic**. In the fermentative type a protein and fat diet should ge prescribed until all discomfort disappears, after which the carbohydrates may be cautiously allowed.

In putrefactive forms it is advisable to prescribe the food in a thoroughly comminuted form. Meats and eggs are usually badly borne, and should be given cautiously. Milk, gelatin, zweiback and cereals are permissible and vegetables only when mashed and strained.

Ulcerative Colitis, Sigmoiditis and Proctitis.—Ulcerations of the intestines may take their origin as a result of acute or chronic colitis developing from infections in abraded areas of the bowel. The infections may be due to the colon bacillus, streptococci or staphylococci or to the organisms producing dysentery or tuberculosis. Of the ulcerations of the colon leading to diarrhea the following are most frequent: entamebic, bacillary, tuberculosis, syphilitic and carcinomatous.

ENTAMEBIC DYSENTERY.—The reader is referred to Vol. IV, page 271, in which all conditions associated with entamebic dysentery are fully described. Since this affection is not an infrequent form of dysentery the entameba should be looked for in all cases of chronic diarrheas. These organisms are best observed in doubtful cases, in the scrapings obtained from ulcerations through the proctoscope. In this affection there are frequent evacuations of the bowels, as many as from ten to twenty a day, and the stools contain mucus, pus and blood. With the passage of these stools the patient is frequently affected with colicky abdominal pains often associated with tenesmus. In addition there are fever, emaciation and exhaustion. For a description of the treatment of this affection, see Vol. IV, page 322.

BACILLARY DYSENTERY.—This affection, which is due to the bacilli of the dysentery group, is accompanied by frequent movements containing blood and mucus; there is noted severe pain and colic in the abdomen, and not uncommonly tenesmus due to the involvement of the rectum. The diagnosis of this condition is usually difficult. The bacillus dysenteriæ may occasionally be found in the stools, and when obtained will agglutinate with the blood-serum. Whenever amebas cannot be discovered in the stools this condition must be suspected, and attempts should be made to isolate the organism. According to Shiga, this disorder is only a subsequent disease in which it is impossible often to isolate the dysenteric bacillus. In this condition the feces are found at times mixed with mucus and the débris from the ulcerated areas. Most frequently, however, the mucus is simply hanging to the feces and is not intimately mixed with it.

Treatment.—The result of treatment of these cases is not always gratifying. **Diet** plays a most important rôle. This should consist mainly of boiled milk for a time, followed by cooked cereals, baked potatoes and vegetables in the purée form and finally scraped beef. In addition to

diet and **rest**, the best results are obtained by means of **irrigations**. Of the remedies to be utilized for this purpose, **silver nitrate solution** (1:500 to 1:1000), **potassium permanganate** and **tannin** are probably the best. Frequent **lavage** of the intestine by means of the duodenal tube with **Ringer's solution** has been of great service in the authors' hands. Internal medication is usually of little help. Of the remedies that may be employed **bismuth** is of some value. In many of these cases occasional mild purges are sometimes helpful. Of these **castor oil** or **liquid petrolatum oil** are to be recommended.

TUBERCULOUS ULCERATIONS.—Tuberculous ulcerations are exceedingly common complications of chronic pulmonary tuberculosis and especially frequent as a late manifestation of this disease. This affection is not always associated with a persistent diarrhea; alternating constipation and diarrhea is not infrequent. Diarrhea is more pronounced when the lesions are in the lower bowel. Pain is the most frequent symptom, occurring usually in the lower abdomen, and it is often most severe before the passage of a stool or after taking food. The stools may be very frequent, and at times may contain small quantities of blood. It is unusual, however, to note the passage of large quantities of blood. Mucus in variable quantities is often admixed with the feces. The finding of tubercle bacilli in the stools is of little diagnostic value, for these organisms are frequently present in patients affected with pulmonary tuberculosis and having these organisms in their sputum. Of great interest are those cases of localized tuberculosis occurring about the ileocecal region as well as those of tuberculous ulcerative colitis. In the former a definite mass is frequently found, resembling a malignant growth. In these cases in addition to the paroxysmal pain, diarrhea alternating with constipation is observed. In several cases of this condition under the authors' care, operation was performed with gratifying results.

In the second variety, *i.e.*, of tuberculous ulceration of the colon, Brown and Sampson have furnished us with a means of diagnosis through the roentgen ray, which has been exceedingly helpful.

Treatment.—The treatment of the diarrhea observed in intestinal tuberculosis is extremely difficult. **Diet** and **rest** play a most important rôle in this direction. **Normal salt solution irrigations** may at times be helpful, but sooner or later it may become necessary to administer opium by mouth or by bowel.

SYPHILITIC ULCERATIONS.—Syphilitic ulcerations of the small intestines are rarely observed. The most common lesion of this disease is in the rectum. When the affection involves the small intestine, the symptoms are those of an ordinary intestinal ulceration; that is, persistent and painful diarrhea remaining unrelieved by the ordinary treatment. When involving the rectum, the picture is so characteristic as to render the diagnosis exceedingly simple. The indurated edges of the elevated ulcer with the smooth base, together with gradually increasing stenosis, is ordinarily sufficiently characteristic to permit of a diagnosis. The finding of a positive Wassermann reaction makes the diagnosis even more certain. In this affection there is usually diarrhea at hand, though at times the stools may be semisolid, and considerable amounts of mucus are usually present and not infrequently shreds of tissues; the appearance of large amounts of blood is rather uncommon.

CARCINOMATOUS ULCERATION.—Carcinoma may be observed in almost any portion of the colon. It may be found in the rectum, pelvic colon, splenic and hepatic flexures, transverse colon, and cecum. Symptoms of incomplete obstruction are among the early signs of this affection, and are manifested by attacks of colic associated with constipation; soon blood or bloody stools occur, which are at first due to congestion, and finally to ulceration. When the disease is in the rectum, there is usually a constant desire for bowel evacuations often accompanied by a discharge of gas with mucosanguineous matter. As the disease advances the contracted area sometimes ulcerates, and constipation may give rise to a marked diarrhea of small watery stools, containing blood and mucus.

FOLLICULAR ULCERATIONS.—This affection is produced as a result of infection in the solitary lymphatic follicles of the bowel. These ulcerations are most frequently observed in the large bowel, sigmoid and rectum and are sometimes so slight as to present the appearance of hardly more than simple erosions. The stools are extremely profuse and persistent and contain small quantities of blood and mucus. The diarrhea is greatly increased by even slight errors in diet, and is extremely difficult to control by almost any form of treatment. Follicular ulcerations are more commonly observed in children than adults.

Treatment.—In the treatment of the diarrhea associated with this affection, **rest** and **carefully regulated diet** are extremely essential. The patient should be placed in bed at rest for a considerable period of time on liquid and semisolid food. In many of these cases temporary relief is afforded by means of transduodenal lavage with **Ringer's solution.** In others **irrigations** through an appendicostomy opening have produced excellent results.

Mucous Colitis.—Mucous colitis is such a well-known affection that but little new can be said concerning it. There are three theories regarding the etiology of this disease; first, that it is entirely a nervous affection and that the mucus formed is purely a nervous hypersecretion; second, that it is due to a catarrh of the bowel, and thus has invariably an anatomic basis; and third, that the disease is due both to a nervous irritation and to a catarrhal condition.

Mucous colitis is always associated with evidences of disturbance of the autonomic nervous system, and there are present the signs of a vagatonia. The symptoms ordinarily noted as signs of this condition are frequent attacks of colicky pains in the abdomen, the expulsion of mucus in the form of a membrane, and usually constipation. While constipation is ordinarily a prominent symptom, diarrhea is occasionally observed in this disease, and may occur during the attacks of colic or when the colic has subsided, and when the discharges of mucus and feces have been passed into the lower bowel, thereby exciting active peristalsis. The liquid movements always contain much mucus in the form of shreds, bands or even complete casts of the bowel. In the treatment of the diarrhea associated with mucous colitis great relief is frequently obtained by the use of **oil enemata**, and the internal administration of **benzyl benzoate** in full doses. **Transduodenal lavage with Ringer's solution** is exceedingly beneficial in many instances.

Simple Colonic Infections.—Among the infections of the bowel that may give rise to persistent diarrhea (not including the amebic and bacillary forms already referred to) are various other microörganisms and parasites that may play an important rôle in this connection. Of these may be mentioned certain intestinal worms, including tapeworm, hookworm, whipworms, flukeworms, strongyloides and various flagellate parasites including lambia, cercomonas and trichomonas intestinalis and ballantidium coli. The diagnosis of the diarrheas and dysenteries produced by these infections can usually be made by careful examination of the stools.

In a study of the diarrheas due to protozoic infection of the bowel Smithies points to a characteristic feature in that there are periods in which normal stools alternate with periods of diarrhea and that the diarrhea appears often suddenly without any apparent cause. The diarrheic stools are evacuated with much gas but without pain. However, it is always observed that abdominal pain or discomfort bears no relation to the movements. The stools are alkaline, often soft, brown or yellow in color, and contain mucus and at times blood. The odor is characteristic, being pungent, ammoniacal and putrid. The reader is referred to Volume IV, which contains a complete study of these affections and the diarrheas produced by these various infections.

Intestinal Obstruction and Stasis.—In intestinal obstruction constipation is ordinarily present. However, when the obstruction is incomplete, alternating constipation and diarrhea may be produced, and this may develop into constipation with complete blocking of the bowel. In association with the diarrhea there is abdominal distention, colic and difficulty and inability often in expelling gas. The stools are small, liquid, offensive and at times contain mucus. As the bowel becomes almost completely blocked, dilatation is noted in the area above the obstruction, and in consequence the diarrhea is increased inasmuch as liquid stools can alone pass the obstructed area.

There are three well-marked varieties of intestinal stasis that may give rise to persistent diarrhea. In the first form the stasis is due to a dilated cecum often in connection with a dilated colon, which is frequently prolapsed, and which retains its contents for an unusually long period of time. On account of the retention, fermentation is produced, which is followed by diarrhea.

In the second form of stasis on account of the prolonged retention, the fecal masses are so channeled that the stools pass through in a diarrheal form. In this condition of fecal impaction or coprostasis often observed in extremely constipated individuals, periods of diarrhea accompanied with abdominal pain and distention are not uncommon. When this irritation due to impaction extends over a long period of time, catarrhal conditions of the bowel are not unusual, and in consequence due to the injury to the intestinal mucosa by the scybalæ and the pressure of the impacted stool, ulcerations may occur, which further increase the tendency to diarrhea.

In the third form of stasis the diarrhea is due to a spastic condition of the bowel giving rise to a frequent expulsion of small round scybalæ. The lower bowel is constantly filled with these masses, which produce an irritability of the bowel, and frequent desire for defecation with the passage of watery stools, containing mucus. In all of these forms roentgen ray, rectal and proctoscopic examinations will quickly clear up the diagnosis.

Chronic Appendicitis.—Although diarrhea is rarely observed as a symptom of chronic appendicitis, the authors are convinced that appendicitis occasionally gives rise to this symptom notwithstanding the fact that these forms of diarrhea are usually attributed to other disturbances such as colitis or disease of the cecum. This condition occurs most frequently when the inflamed appendix is plastered against the bowel. In one of the authors' cases the nature of the trouble was revealed by the roentgen ray. There were no symptoms definitely relating to the appendicitis, except the diarrhea. In another case the patient developed an acute attack of appendicitis. The appendix was removed, and there were no further symptoms.

Diverticulitis Involving the Sigmoid and Rectum; Polyposis of the Colon.—Diverticulitis is associated in its earlier stages with constipation in addition to abdominal discomfort and a general distention from gas; after a time, however, the constipation not uncommonly alternates with diarrhea and the pain becomes localized in the region of the sigmoid, producing the symptoms much like those of appendicitis, but on the left side. The diarrhea observed in this affection is of the coprostatic type, that is, the lower bowel is filled with hard fecal masses, which produce an irritability of the bowel, and frequent desire for defecation. On account of the prolonged retention the fecal masses may be so channeled that the movements pass through in a liquid state.

Multiple polypi of the colon frequently give rise to severe diarrhea and profuse hemorrhages. The movements are watery and usually contain blood and mucus. When the disease invades the rectum, which is not infrequent, the diagnosis may be confirmed by proctoscopic examination.

Lesions of the Brain and Spinal Cord.—In certain affections of the brain and spinal cord, involuntary liquid stools occur, due to disturbed neuromuscular function of the colon, and more especially the rectal sphincter. Paralysis of the sphincter may occur in diseases of the brain often associated with loss of consciousness. It is also observed at times as a result of spinal cord lesions.

Diarrhea of central origin is frequently noted in cerebral hemorrhage, brain tumors, locomotor ataxia and transverse myelitis. The symptoms relating to the bowel movements in these conditions vary with the severity of the involvement. The slightest pressure exerted by the patient will be followed by the passage of liquid stools; a cough or sneeze may bring about a similar result. In some instances the movements may pass when the patient urinates or when he walks; in others he may be unaware of the bowel discharge, the stools passing unconsciously in liquid form.

Disturbances of the Glands of Internal Secretion.—The disturbances of the endocrine glands need only be considered here in so far as they are concerned in the production of diarrhea. The diarrhea noted in hyperthyroidism has been explained on the basis of a simple hyperperistalsis. It is at times observed as an early symptom in this affection before other symptoms appear, although it usually occurs as a late manifestation. It is quite probable that many cases of so-called nervous diarrhea on careful examination will be found to belong to this class. In the pancreatic forms, large fatty stools, usually soft, but occasionally diarrheal, aid us in reaching a correct diagnosis. In the suprarenal forms the diarrhea is due to a disturbance of the internal secretion of this gland producing a hyperperistalsis. The intestinal secretion is not ordinarily disturbed in this affection, though at times the stools may contain an excess of fat and mucus.

It is quite possible that disturbances of the internal secretions may provoke changes in the intestinal secretion at times, which in turn may form the basis for fermentation and consequent diarrhea. In the treatment of these forms of diarrhea Kast advises, especially in those due to hyperthyroidism, the administration of lukewarm water enemata containing from 10 to 15 drops of **adrenalin**; this is said to cause immediate cessation of this symptom.

Diarrhea Due to Cardiorenal Disease.—Diarrhea is frequently observed in cardiorenal conditions. In myocardial insufficiency due to the general passive congestion of the abdominal organs diarrhea is a common symptom. Diarrhea of nephritic origin is probably due in the larger proportion of cases to the excretion into the bowel of irritating toxins, as is shown by the fact that the severity of the diarrhea varies with the exacerbations of the nephritic disease. In patients affected with chronic nephritis extending over a long period of time uremic ulcers have been observed in both the small and the large bowel. In some cases of chronic nephritis severe and even fatal diarrhea may develop.

Diarrhea Associated with Disturbed Metabolism.—Diseases associated with disturbed metabolism such as pellagra, sprue and gout are often accompanied by diarrhea.

In pellagra the diarrhea is usually very severe, and is accompanied by pain. It may alternate with constipation. The stools are either serous or bloody and often contain undigested food elements, especially when the gastric and pancreatic secretions are disturbed. In the late stages of severe cases, an uncontrollable diarrhea occurs which is a great factor in causing loss of strength and weight. In the treatment of the diarrhea due to this disease, **rest in bed** and a **diet** as nutritious as possible should be given. **Astringents** and **arsenic,** and **starch** and **opium enemata** are useful in controlling the diarrhea. For a full description of this disease see Vol. IX, page 205.

The diarrhea in sprue is very characteristic. At first it is accompanied by pain and tenesmus, the stools being liquid and dark; later the movements occur usually in the early part of the day, and are unattended by pain or tenesmus. The sprue stool is fatty, copious, frothy and fermented, light in color and acid in reaction. P. H. Bahr, Brown, Pratt and Spooner have found an absence of pancreatic ferments in the stools. In addition to the pancreatic insufficiency, ulcerations of the colon may also play a part in the causation of the diarrhea. In the treatment in addition to **rest** and the usual **dietetic régime** satisfactory results have been obtained by Brown by means of the internal administration of large doses of **pancreatin.** The reader is referred to Vol. IV, page 173 for a complete description of this affection.

Gout is more frequently accompanied by constipation than diarrhea; when loose movements prevail, other gastro-intestinal symptoms may be present, such as foul breath, furred tongue, pyrosis, flatulency and abdominal pain. In addition to the usual treatment of gout, the gastro-intestinal manifestations should be treated symptomatically.

Diarrhea Following Operation on the Gastro-Intestinal Tract.—CHOLECYSTECTOMY.—In a small group of instances the operation of cholecystectomy is followed by a persistent diarrhea and very rarely this condition may even occur after a simple cholecystotomy. In all of these cases there is present according to the authors' experience a diminution or absence of pancreatic ferments as is revealed by a fractional analysis of the duodenal contents according to the Einhorn method. The exact nature of this form of diarrhea is unknown, but it is quite possible that it may be due largely to the disturbed pancreatic secretion. According to Brown, relief is afforded almost at once in these cases by means of pancreatin and lime salts.

GASTRO-ENTEROSTOMY.—Gastro-enterostomy is not infrequently followed by an intractable diarrhea. This condition may appear almost immediately following operation and persists for a considerable length of time. The cause of this form of diarrhea has been explained on the basis that due to the rapid passage of undigested food into the bowel, mechanical irritation occurs to such a degree as to lead to the production of the liquid movements. In some of these cases the activity of the pancreatic secretion is impaired which may also result in intestinal fermentation accompanied by diarrhea. In this condition the stools are frequent, very watery, and contain many undigested food remnants. Diarrhea following this operation appeared in 8 per cent. of the cases reported by Finney and Friedenwald. The authors have found it advisable to regulate the diet immediately following this operation, and to continue for several months this plan which has proved very satisfactory in their hands.

PYLOROPLASTY.—Like gastro-enterostomy, pyloroplasty is at times followed by diarrhea, however, never of so severe a type as one finds after gastro-enterostomy. Diarrhea after operation appeared in 5 per cent. of the cases of Finney and Friedenwald, mainly, however, after the first week or ten days, and it was frequently due to errors in diet. Inasmuch as the operation is performed in most instances upon patients affected with pyloric obstructions which have been troublesome often for as much as a year, it is easily understood how on account of the rapid passage of the gastric contents through the stomach a weakened intestine may be overtaxed and intestinal indigestion and diarrhea be induced. On this account, as well as because the stomach has in most instances temporarily lost its tone, and inasmuch as in some instance portions of ulcers have not been entirely resected, Finney and Friedenwald have found it advisable to regulate the diet immediately following operation and to continue this plan for some time. Since these restrictions have been carried out diarrhea has been rarely noted. The diarrheal stools following pyloroplasty present similar characteristics to those following gastro-enterostomy.

APPENDECTOMY.—Attention has already been called to the fact that in a small group of cases chronic appendicitis is accompanied by diarrhea which is overcome by appendectomy. In a smaller group the removal of a chronic appendix may produce a similar condition. The cause probably lies in some injury to the adjacent bowel occasioned by operation. The diarrhea in this condition may be profuse, and may continue on for a considerable period of time following the operation. It can usually be corrected by means of **rest** and **diet.**

RESECTION OF THE BOWEL.—One not uncommonly observes diarrhea following bowel resections, the cause of which is not difficult to explain if the severe injury inflicted upon the intestines by the operation is taken into consideration. The movements may be exceedingly frequent and profuse, leading to such intense exhaustion and weakness as to even render the condition of the patient alarming. In most instances by means of **rest, restricted liquid diet** and the administration of **opiates** this condition may be overcome.

Nervous Influences.—By means of examination of the functional activity of the intestine as well as by careful investigation of stools nervous diarrhea can now be differentiated from diarrheas due to organic disease. These forms of diarrhea have their onset often after severe excitement, worry or shock. Nervous diarrhea is produced by increased hypermotility of the bowel, the stimuli producing this affection being either psychic or reflex; or there may be a combination of both forms. The psychic variety is produced by worry or shock, while the reflex form occurs as a result of bodily stimuli as is noted for example after sudden chilling of the body. The onset of this condition is frequently quite sudden, associated with a gurgling sensation in the abdomen, while the patient is still enjoying good health. The stools are quite liquid in character, and are accompanied with but little discomfort in the abdomen. Between the attacks, the patient experiences but little discomfort, and can partake of all varieties of food with impunity. The attacks recur at shorter or longer intervals, at times so frequently as to render the patient extremely weak. In addition nervous and neurasthenic symptoms frequently develop. On examination of the stools no abnormal constituents are ordinarily revealed, and there is but little fermentation at hand. On account of the increased hypermotility, undigested food remnants may be discovered at times.

Inasmuch as dietetic measures and the administration of drugs play an exceedingly unimportant rôle in the relief of these cases attention must be directed to other measures. Of these **psychotherapy, change of scene, cold sponges,** and **baths** exert a far more beneficial effect. The diet should not be too greatly restricted and must be directed to a great extent to the building-up process. In intractable forms of this affection, **rest cures,** extending over long periods of time (eight or ten weeks), have been of great service in the treatment of the authors' cases.

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CHAPTER XXV

THE ACUTE DIARRHEAS OF EARLY LIFE

By John Ruhrah, M.D.

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Acute ileocolitis, p. 711:—Definition, p. 711; Symptomatology, p. 711; Catarrhal form, p. 711; Ulcerative form, p. 711; Membranous form, p. 711; Diagnosis, p. 711; Treatment, p. 711; Prognosis, p. 711; Pathology, p. 712.

Definition.—Diarrhea is a term used to designate frequent loose movements of the bowel. Like vomiting it is a symptom which occurs in many diseases and is due to many different causes.

The term diarrhea covers a multitude of conditions for which there is as yet no adequate classification, since the clinical manifestations rarely coincide with the bacteriological and pathological findings. A classification into simple diarrhea and intoxications and infections seem to be simplest and practical. The toxic and infectious diarrhea may be described under the headings of acute gastro-enteritis (including cholera infantum) and acute ileocolitis.

Etiology.—The predisposing causes of diarrheas are known but not understood. Heat is the first factor; diarrheas are all more frequent in warm climates and during hot weather. The sexes are about equally affected. Age is a big factor and most of the severe diarrheas occur in early childhood, especially during the first two years. Certain children are more susceptible and one attack weakens the resistance of the intestines and thereby predisposes to subsequent attacks. Diarrhea occurs most frequently in the children of the poor who live in overcrowded, badly ventilated, unclean homes and are not always able to be provided with suitable food, clean milk and ice wherewith to preserve it. Diarrheas are frequent in institutions where large groups of children are improperly fed and cared for.

The toxic and infectious diarrheas are associated with the ingestion of toxins developed in foods and with various bacteria. Just how much is dependent upon each factor in a given case it is impossible to say. Comparatively little is known about the toxins which cause diarrhea. Of the bacteria somewhat more is known, but our knowledge is far from complete. In some cases specific bacteria are the cause, such as the Shiga-Flexner dysentery bacteria, while in others there are various forms of streptococci and other organisms. Diarrhea as ordinarily seen is not due to any specific organism but to mixed infection. This may be taken in with the food or may be present in the intestines and started into activity by bodily changes brought about in some way with very hot weather. Certain cases seem to come from unfit water or ice, some from foods other than milk, but the majority of the cases are associated with impure milk. The disease may at times be distinctly infectious and be conveyed from one child to another by direct contact, or by the hands of the nurses or by flies feeding upon the exposed stools and then walking over the nipples of bottles, or getting into uncovered milk.

The relation of diarrhea to bottle feeding is important. The figures vary but it is safe to say that from 95 to 97 per cent. of the cases occur in bottle-fed babies, and most of these babies fed on spoiled cow's milk. Green or over-ripe fruit, foods unsuited to the child's digestion, may start a simple acute intestinal indigestion which under certain not very well understood conditions may change to a severe or even fatal toxic or infectious diarrhea. Of all the foods, however, dirty impure milk is the most frequent cause.

In the simple diarrhea there is no toxic or infectious material as a cause. In this the causes are: mechanical, such as follows the ingestion of green fruit; medicinal, from laxative drugs; reflex, from nervous conditions, resultant from fright, overheating or chilling; and elimination, such as may be seen in nephritis.

Symptomatology.—Acute intestinal indigestion may occur either with or without gastric involvement. If the stomach is involved as well there will be the characteristic symptoms of gastric indigestion. In infants and young children this is a very frequent disorder usually due to errors in diet but sometimes caused by exposure to heat or cold.

The most salient clinical features are pain and diarrhea. If the stomach is involved vomiting is the rule, although it may be absent in some cases. There is usually distention of the abdomen and almost always fever, which in many cases is alarmingly high. The stools are increased in number, the first one usually consisting of normal feces but loose, and later the movements are thin, discolored, green or brown, and usually have a decidedly objectionable odor. Mixed with the stools are particles of undigested food, largely undigested food or fat and generally there is a large quantity of mucus and sometimes thin watery fluid.

Diagnosis.—The diagnosis from the infections and intoxications is made by the comparative mildness of the attack and its short duration. At the outset there is no way to tell what form of diarrhea one is to deal with so that if treatment is instituted in the first twenty-four hours it should be exactly the same as recommended for the infections and intoxications with the exception that the return to the regular diet may usually be made quite rapidly.

ACUTE GASTRO-ENTERITIS

Definition and Etiology.—This covers a large group of diarrheas seen throughout the year but most frequently in summer. They are probably due to a large number of contributing causes with either bacteria or bacterial products as the immediate cause of pathogenic changes. A very large number of names have been used for this condition and all sorts of classifications have been attempted but they seem to have but little practical value. The most common terms used in addition to the one chosen are summer diarrhea, summer complaint, acute intestinal intoxication, and cholera infantum. This last term perhaps is best limited to a certain small number of cases in which the large number of watery stools containing small flakes of epithelium suggests the rice water stools of Asiatic cholera; hence the name. The predisposing causes are as given at the beginning of this article. The disease is chiefly seen in hot weather. Nearly all of the cases and deaths are in bottle-fed babies and it is by far the most prevalent among the poor and over-crowded and in children under two years of age.

Symptomatology.—The symptoms of the disease usually begin very shortly after infection, after the ingestion of food containing toxins, or after exposure to extreme heat. In the large majority of cases the onset is sudden. The child is taken with an attack of vomiting and with diarrhea which begins simultaneously or shortly after. In other cases the severe disease may follow some neglected gastric or intestinal indigestion which after several days or weeks lessens the resistance of the intestinal tract and makes way for the establishment of a severe diarrhea.

VOMITING.—The vomiting may disappear after a few hours or it may persist and form one of the most troublesome features of the disease. The vomited matter consists usually of milk, sometimes large quantities of clear fluid and at other times bile-stained mucus. In the severest cases, with paralysis of the bowel, the vomiting may take on the character seen in intestinal obstruction and be even fecal in character. In the majority of cases vomiting after the first few days is not so troublesome and often depends largely upon the amount of care used in feeding the child, too much or unsuitable food causing a return of the trouble.

DIARRHEA.—The diarrhea is always a marked feature but is exceptionally variable. The stools are usually frequent, although occasionally very severe cases may be seen in which only one or two very bad stools are passed in twenty-four hours. If the disease begins suddenly the natural intestinal contents are first passed and then large quantities of mucus mixed with fecal matter. The stools are usually greenish or brownish in color, with a musty odor of mucus and at other times with the odor of putrefying flesh. The stools may contain pus and blood. Occasionally fair sized hemorrhages may occur but more often the blood is limited to streaks or to masses of blood and mucus mixed together. The number of stools is not of as much importance as their character, with the exception of the cases in which large fluid stools are passed at frequent intervals. In many instances there is a very marked chafing of the buttocks and thighs, sometimes this may be so severe as to extend through the skin in certain places.

LOSS OF WEIGHT.—The loss of weight is very great, sometimes 8 to 10 ounces a day and sometimes very much more than this so that in a few days the child's appearance may change so much that one would no longer recognize it.

TEMPERATURE.—The temperature varies. In some cases there is little or no elevation while in others there is hyperpyrexia, sometimes temperatures of 106°, 107°, or 108° F. (41.1°, 41.6°, or 42.2° C.) being noted. These cases with high temperature are attended with very marked prostration, the child usually becoming semicomatose or even profoundly unconscious. The skin is pale or cyanotic and in many instances the child feels cold or clammy to the touch and the mistake is made of applying heat. In all diarrhea cases the temperature should be taken in the rectum. Children with a very high temperature may die from the hyperpyrexia but if hydrotherapy is promptly instituted some may recover. The average case runs a more or less irregular fever varying from normal or below to 103° or 104° F. (39.4° or 40° C.). Usually the temperature and the general condition of the child coincide, but this is not true of all cases, as many afebrile attacks may be of great severity or even fatal, whereas many cases with high prolonged temperature may make a perfectly good and often rapid recovery. Younger children at the beginning of the attack are usually restless and also carry a moderately high temperature. With a hyperpyrexia the child quiets down as noted above.

In many cases, especially if they last several days, the child becomes listless and drowsy and does not want to be disturbed; it cries and is irritable when it is handled or when attempts are made to feed it or to administer medicine. Some infants give evidence of being in severe pain and older children often complain of colicky pains in the abdomen. The examination of the child shows more or less evidence of a loss of weight. There is much gas in the intestine, the abdomen is distended, sometimes very tense, but in other cases in which the gas is not a feature the abdomen may be relaxed and assume a more or less boat-shaped character as described in tuberculous meningitis. There is often tenderness on palpation.

CHOLERA INFANTUM.—The type of disease known under the name of cholera infantum probably has nothing special in its etiology but is merely the reaction of a very badly poisoned body. These cases resemble in many ways Asiatic cholera and are characterized by a very severe toxemia, high fever, frequently hyperpyrexia, severe and continuous vomiting, large frequent stools which consist very largely of a fluid containing small flakes of epithelium and some mucus, suggesting the rice water stools of true cholera. The loss of weight is very great and very rapid and the blood of the patient often becomes thickened unless water is supplied subcutaneously or by one of the other methods. Most of these cases prove fatal; occasionally one will recover, often when least expected.

Diagnosis.—The diagnosis of acute gastro-enteritis presents but little difficulty. The vomiting and frequent stools are self-evident. Whether one is dealing with a simple or a toxic diarrhea cannot be definitely told at the outset, although one with a large clinical experience can frequently surmise the nature of the disease. A diarrhea coming on in a child entirely breast-fed with little or no fever, no toxemia, is likely to turn out a simple matter although not necessarily so. In the simple diarrheas the mere cleaning out of the bowel with twenty-four hours' rest is sufficient to cause the symptoms to subside either largely or entirely. An error very frequently made is the over-looking of the presence of some disease because there happens to be a diarrhea at the same time; but the most common error is the failure to determine a pneumonia or one of the acute infections, such as scarlet fever, which may occasionally start with loose bowels.

In severe diarrheas there may be the syndrome suggesting meningismus, or as some authorities call it, serous meningitis. Meningeal symptoms are not infrequent, particularly in the severer forms of the disease. They consist very largely of slight rigidity of the neck and a slight rigidity of the muscles of the extremities; but this rigidity is not of the steel-bar variety seen in true meningitis. The patient's head can usually be flexed if firm pressure is continued for a short time. If the fontanel is open it is found to be sunken rather than raised. Some of the cases test the diagnostic ability of the physician and in cases of doubt a lumbar puncture should always be resorted to.

Treatment.—PROPHYLAXIS.—Much can be done in preventing the disease by having the **child in the fresh air** as much as possible, protecting it from fatigue or overheating, **keeping it clean, cool and comfortable.** Another important factor is to see that the child during the heated term is given **additional water to drink,** particularly if the appetite is not good and the child is not taking sufficient water with the fluid foods,—in other words more water and less food during hot weather. The removal of the child from a crowded city to the country or to cooler climates during the heated term is important but can be applied to but few of the population.

MILK SUPPLY.—The chief feature in preventing diarrheas is the securing of a pure milk supply, particularly during the heated term. It is better to use **pasteurized or sterilized milk** unless the fresh milk is produced under ideal conditions and kept cold from the time it leaves the cow until it is consumed by the baby. Inasmuch as this cannot be done, particularly by the poorer classes of people, the author is becoming more and more convinced that diarrheas will be prevented in the future by using during the heated term one of the **dry milks or milk derivatives,** instead of fresh milk. Other milks which can be used are **condensed milk mixtures,** and the **malted milks** if the latter do not cause too frequent stools. **Cows' milk which is sold loose should never be used for infant feeding.**

The danger of scurvy is negligible and where condensed milk and malted milk are used small amounts of **orange juice** will act as a preventative. In very hot weather a child should be under-fed rather than over-fed, and all articles of food of which there can be any question whatever are best withheld.

GENERAL MANAGEMENT.—The general management of cases of diarrhea is important but is somewhat difficult to detail inasmuch as the circumstances under which a diarrhea may occur are so varied. Common sense must be used in all cases. The general principles are to **put the child at rest**, have it disturbed as little as possible, have as few people see it, handle it and talk to it as is possible. In nervous children this cannot be too highly insisted upon as many a child is killed by an over-anxious family and friends trying to do something. In the treatment of diarrhea it is always well to remember Locke's dictum, "It is better to do nothing than to do amiss." The child should be put in the coolest room in the house if the disease occurs in summer and if it is possible to have it removed from the hot city to a cooler place this is of very great advantage. The prompt removal to some of the summer homes or hospitals, many of which are to be found within easy reach of most large communities, is to be recommended particularly where the home surroundings are poor and the nursing apt to be insufficient and unintelligent.

The first thing to do in a case of diarrhea is to **empty the stomach and bowel and give them rest for at least twenty-four hours**, supplying in the meantime sufficient fluid, in the form of water by mouth, or in severe cases a salt solution as noted below. In severe cases where the vomiting is continuous the stomach may be washed out with a 4 per cent. solution of **sodium bicarbonate**. The same treatment may be used for the rectum and lower bowel. **Castor oil** may be given in full doses, or, if this is not retained, **sodium sulphate** in solution flavored with a little **aromatic elixir or calomel** in one-tenth grain (.00648 gram) doses every twenty minutes until one grain has been given. If there is evidence that the gastro-intestinal tract has been thoroughly evacuated, these measures are best omitted.

DIETETICS.—The diet in all diarrheas is one of the most important parts of the treatment and one of the most difficult. In exclusively breast-fed children, after the initial purge, it is usually a good plan to give nothing but plain water until the nature of the diarrhea is apparent. The flow of breast milk can be maintained by the use of a breast-pump or by manual expression. In cases where there is no vomiting the child may usually be put to the breast for a few moments and immediately given either plain water or barley water to make up the difference required. If there is vomiting the breast should be withheld and nothing given except plain water until the stomach is quiet. If the vomiting is severe, **washing out of the stomach with a solution of bicarbonate of soda**, one dram (3.9 grams) to the pint (.57 liter) is to be recommended. If the bowel condition becomes normal promptly, the breast-feedings may be resumed. It generally is a good plan to give a small amount of water for several days so as to dilute the milk and to lessen the amount taken.

In infants who are partly bottle-fed and partly breast-fed and in those entirely bottle-fed it is important to arrange the diet so that it will meet the requirements of the case should it turn out to be one of the severe variety. This means stopping all nourishment for twenty-four hours, giving nothing but plain water or a little thin barley water or a little very weak tea. At the end of twenty-four hours, if the stomach and bowel have been thoroughly emptied and given a rest and there is no vomiting, partly breast-fed child may usually be allowed to take breast milk at the usual feeding intervals along with water before the feeding, and the nursings may be increased according to the condition of the stools. Then **barley water** or **rice water** may be used. An error which is ordinarily made is to fail to starve the child boldly during the first twenty-four hours and the second error is to starve it too long.

The proper feeding in severe diarrheas is a difficult question and one about which there is a great deal of difference of opinion. The history of the feeding in diarrheal diseases is an interesting though somewhat tiresome chapter and all the various food components have been praised and condemned many times. The truth of the matter is that we do not know sufficient about the feeding of these cases to speak very dogmatically concerning them. Many years of experience leads one to think that Jacobi was wise when he said that "some make the same mistake over and over again and call it experience."

There are numerous methods of feeding, some of the most important of which will be outlined. The author believes that the only food component which should be avoided in all cases is fat. Children with diarrhea certainly do better on either **fat-free** or **foods** relatively low in fat.

The first thing in the diet of the acute diarrheas is starvation for the first twenty-four hours, allowing only water or a small amount of weak tea or sometimes a small amount of thin barley or rice gruel. At the end of twenty-four hours it is important to begin some sort of nourishment in the child providing there is chance of its being retained and digested. It should always be remembered that many of the children with diarrhea actually starve to death,—"they die cured." One should determine the line of feeding to be tried and if not successful a change to some other method should be made. But unless one is certain that the method in use is not going to succeed, changes should not be made too rapidly.

In cases in which there is much fermentation and gas, perhaps the best method is to use **protein milk** or one of its modifications.

The author prefers skimmed milk mixtures to which **calcium caseinate** has been added in the proportion usually of one-third of an ounce (10.37 grams) to a pint (.57 liter). The proportion of skimmed milk may be varied to suit the patient but ordinarily half milk and half water with the calcium caseinate added and then boiled five minutes gives a satisfactory mixture. It should be stirred constantly while being heated. If the calcium caseinate is dissolved in about one-third of the mixture while cold and the remainder of the milk heated and then the cold solution poured into the hot milk and boiled, a smooth mixture will result. This should be used after the manner of protein milk, that is, start with very small amounts, one dram (3.9 gram) for instance, and double the size of the feeding each time providing this amount is retained.

The character of the stools should change to the so-called soap stools and when this happens some form of **carbohydrate** should be added to increase the calories in the food. **Sugar** is usually added beginning at one per cent. or **dextrimaltose** may be used and later barley gruel may take the place of the plain water. The protein milk as suggested by Finkelstein is made as follows:

Heat one quart of whole milk to 100° F. (36.66° C.), add four teaspoonfuls of essence of pepsin, and stir. Then let the mixture stand at 100° F. until the curd has formed. Put the mass in a linen cloth and strain the whey from the curd. Remove the curd from the cloth and press it through a fine sieve two or three times, using a wooden mallet or spoon. While doing this one pint of water should be added. The precipitate should be very finely divided and the mixture should look like milk.

For those who cannot afford casein milk Reuben suggests the following:

Add two junket tablets to a pint of milk. After it has stood for one-half hour strain, discard the whey and rub the curd through a fine sieve together with a pint of milk and then add enough plain boiled water to make one quart (1.14 liter.) The whole mixture is then brought to a boil with constant stirring and is then ready for use. This has nearly the value of casein, but lacks the acidity, a point which does not seem to be essential.

It should be remembered that protein milk feeding is starvation diet and as soon as the stomach and intestinal tract will stand it the food should be increased to cover the needs of the child in question. The failure to remember this is the cause of many of the late mishaps. If the **old-fashioned buttermilk**, that is the milk left after churning butter, can be obtained it is a most useful food either diluted with water or with barley water, half and half, and given in the same way as the protein milk.

Another method of feeding is an old Dutch one, for the details of which the author is indebted to Dr. H. L. K. Shaw of Albany, N. Y. This method sounds astounding but will give good results. It follows:

Raw milk with the cream left in is generally used although some or all of the cream may be removed to advantage in severe diarrhea. It is very important that the milk be unheated. The milk is placed in a bowl and is allowed to stand for twenty-four hours or until it is like junket. It is then churned for five minutes. One heaping tablespoonful of granulated sugar and flour each are mixed with a little of the churned milk. Then the rest of the milk is added and boiled for five or ten minutes, being stirred constantly. If in the cooking it appears to be separating, a little more flour is added. This milk is kept in a room temperature, not in a refrigerator, and should be made two or three times a week. It is given at room temperature without heating. The first feedings should be small and diluted with water and after the first day or two the full strength milk may be used.

Another method of feeding which is much in vogue and which has much to recommend it in cases in which there is putrefaction or foul stools, or where the child is losing fluid rapidly from the tissues, is the use at the beginning of cereal gruels, such as barley gruel or rice gruel.

It is usually made in the proportion of one level tablespoonful of flour to a pint of water. A pinch of salt and enough water to make up the pint is added and the mixture is boiled for fifteen minutes. This may be sweetened a little either with saccharin or with dextrimaltose. After a day or two the skimmed boiled milk or one of the dry milks may be added to this, a level tablespoonful of either of the above being used to four ounces of gruel and then increased until it is either in the proportion of three tablespoonfuls to four ounces or in some instances in older children four tablespoonfuls to four ounces.

Other methods of feeding during the acute stage, particularly in those where there is putrefaction, consist of the use of sugar mixtures such as one level tablespoonful of dextrimaltose to four ounces of water, starting with a teaspoonful or two and increasing the amount if the child bears it well and then eventually adding skimmed boiled milk to the mixture. The sweetened condensed milks are sometimes used with good results, starting with one teaspoonful to two ounces of boiled water and increasing to the strength of one teaspoonful to one ounce of boiled water. Malted milk is useful in some instances and may be given in the strength of one level teaspoonful to an ounce.

Nothing in the practice of pediatrics requires such nice judgment as the return to the ordinary diet. One with experience will generally do it skillfully, but the reasons for making or not making the changes are so complex that they defy description.

MEDICINAL MEASURES.—The drug treatment is important but the results obtained are often unsatisfactory. Countless drugs have been recommended. There are several which have stood the test of time.

Opium should be discussed first. It is at once a most valuable and most dangerous remedy. Opium or its derivatives should never be used in diarrhea without considering whether toxic material may be retained in the bowel or not. If there is doubt do not give it. In simple diarrheas where there is pain or frequent stools it gives prompt relief. In the other forms where there is pain or too frequent stools or straining one may give it tentatively, always starting with a minimum dose and feeling the way with larger doses. In severe cholera infantum with great losses of fluid, **morphin sulphate** may be administered hypodermatically. The author's own preference in other cases is **codein sulphate** or **paregoric.** If the toxemia becomes more pronounced after using such drugs, omit them. It is easy to check a diarrhea with a large dose of opium but the child may die from the absorption of retained poisonous material that were better excreted.

Bismuth preparations may be used. The most effective is **bismuth subnitrate,** but as it is often desirable to give bismuth in connection with alkalis it is safer to use the **subcarbonate** and so avoid the danger of nitrite poisoning which may occur from the subnitrates breaking down in the presence of an alkali. Bismuth and **prepared chalk** is the frequent combination; five or ten grains (0.324 or 0.65 gram) of the chalk is added to each dose. Large doses are best; 5 grains (.324 gram) to a dram (31.10 grams) may be given every two, three, or four hours. Twenty to thirty grains (1.3 to 1.95 grams) are usually given at a time. More astringent and more powerful as an antiputrefactive agent is **betanaphthol bismuth** which may be given in powders of from 1 to 5 grains (.065 to .324 gram).

Tannic acid derivatives such as **tannigin** or **tanalbin** may be used in from 1 to 5 grain doses in powders. They pass unchanged through the stomach and tannic acid is liberated in the alkaline intestinal contents. When these preparations are being used, there should be no alkalis in the food or otherwise lest the tannic acid be set free in the stomach and cause vomiting.

Fullers' earth in water with or without astringents and chalk is often used. The following prescription has been found of great use by the author.

℞

Prepared chalk	ʒ ii	(7.8 grams)
Fuller's earth	ʒ iv	(15.50 grams)
Tincture of kino	ʒ iii	(11.25 c. c.)
Oil of anise	♏ iii	(.18 c. c.)
Water to make	℥ iii	(89 c. c.)

M. Sig.—Shake well. Teaspoonful every two (or three) hours.

Cultures or tablets of **lactic acid bacilli** or the so-called **Bulgara bacillus** or **bacillus acidophilus** have had quite a vogue, the idea being that the pathogenic organisms are driven out of the intestine by changing the intestinal flora. Sometimes this method gives good results but the author rarely uses these agents.

In persistent diarrhea sometimes the administration of acid, such as **aromatic sulphuric** or **hydrochloric acid**, gives remarkable results.

Saline Injections.—In cases where large quantities of fluid have been lost, **subcutaneous, intravenous, or intraperitoneal injections of normal salt or Ringer's solution** may be used, from 1 to 8 ounces (30 to 236 c.c.) or even more, according to circumstances.

Intravenous injections may be used when there is need of haste in getting fluid into the body. In small infants the femoral vein may be used, or if the fontanel is open the superior longitudinal sinus. This is from 2 to 5 mm. under the skin and is wider and deeper posteriorly.

In intraperitoneal injection the skin and subcutaneous tissue are picked up between the thumb and forefinger and the needle introduced in an upward direction through the abdominal wall. The midline just below the umbilicus may be chosen. Do not enter a distended bladder. The warm solution may be run in from an infusion bottle; from 100 to 250 c.c. in young children and from 300 to 400 c.c. in older children may be given every twelve to twenty-four hours, if necessary.

Prognosis.—The outlook in cases of diarrhea is important. Most of the cases and most of the fatalities occur in bottle-fed babies under two years of age, and in considering the first two years a greater proportion is seen in the first year. The prognosis is based upon the surroundings and the care which the child will receive, on the physical condition of the child itself, and on whether the diarrhea is a simple or a toxic one. Weak children in poor surroundings with little or no care usually do badly and generally die. Strong children in good surroundings who can be properly cared for have a better outlook but even many of these succumb. If the weather is very hot and humid the child is more apt to do badly than if the weather is cold and dry. Death generally takes place in coma or during a convulsion and stupor; twitching or actual convulsion occurring after the onset of the disease is to be regarded as of grave import. Hyperpyrexia, especially when difficult to control and long-continued, is another dangerous symptom. Persistent vomiting, the occurrence of nephritis, the persistent loss of fluid through large liquid stools are also bad. Children whose tissues become like dough to the touch always die.

Pathology.—The bowel contains mucus and fecal material similar to the stools. There may be some congestion of the mucous membrane and some swelling of the lymph-nodes. There are degenerations in the epithelium. There may be an associated nephritis, bronchopneumonia, and degenerations in the liver cells.

ACUTE ILEOCOLITIS

(Enterocolitis; Enteritis; Dysentery; Inflammation of the Bowels)

Definition.—This disease is an inflammation of the large and small bowel characterized by frequent movements, tenesmus, and marked constitutional disturbances. Three forms may be described: catarrhal, ulcerative and membranous.

It is often impossible to distinguish cases of acute gastro-enteritis from cases of ileocolitis and the two may occur together. In ileocolitis the lesions are lower down and involve the large bowel as well. In many cases the Shiga-Flexner bacillus can be demonstrated.

Symptomatology.—CATARRHAL FORM.—In this form there is usually a sudden onset with vomiting and diarrhea; the stools contain blood and mucus. There is great pain with tenesmus. The stools are very frequent. There are fever and toxic symptoms with more or less prostration. In mild cases the acute symptoms last about a week, but there is a great tendency to become chronic. In severe cases the acute symptoms may last weeks.

ULCERATIVE FORM.—This form is usually secondary to a gastro-enteritis. There is not so much temperature as in the preceding, and the stools are not as frequent. Blood is seen sometimes. There is progressive emaciation with great weakness. The diagnosis is made from the general history.

MEMBRANOUS FORM.—This form is always severe. There is a sudden onset with vomiting and high fever as in the catarrhal farm, however, usually more intense. There may be marked nervous symptoms. The diagnosis is made on the presence of pieces of membrane in the stools. Otherwise the symptoms are like those seen in catarrhal ileocolitis.

Diagnosis.—Acute ileocolitis is differentiated from typhoid by the Widal reaction and by the slower invasion of typhoid; and from intussusception by the constipation following the onset and the symptoms of obstruction. The membranous form is sometimes mistaken for meningitis if the cerebral symptoms are marked. Diarrhea is rare in meningitis.

Treatment.—This is very much the same as in acute gastro-enteritis. **Fresh air or a change of air** is important. **Opium** is needed for the pain and frequent stools. **Irrigation of the bowel** is also useful. This is best accomplished by flushing the bowel with **normal salt solution** and then using **fluid extract of witch-hazel** (1 dram to a pint), or some other astringent. **Nitrate of silver** is sometimes useful.

Prognosis.—This is bad in all cases. Recovery is attained in many of the catarrhal forms, but relapse is frequent. The ulcerative form is usually fatal, but sometimes partial recovery takes place, and the child may die of some inter-current affection. The membranous form is usually, though not always, fatal. In the poor, in previously ill children, and in hot weather the outlook is especially bad.

Pathology.—The lymph follicles are enlarged and ulcerated in most cases. In others there is a simple catarrhal inflammation and there may be simple ulcerations of the mucous membrane. In other cases there is a membranous inflammation. The lesions are for the most part in the colon and lower part of the ileum. The most frequent complicating lesions are bronchopneumonia and nephritis.

CHAPTER XXVI

DISEASES OF THE LOWER COLON AND RECTUM

By Jerome M. Lynch, M.D., F.A.C.S.

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Examination and Diagnosis.—Much can be learned from the history of a patient; and when this information is combined with that obtained from a digital and proctoscopic examination, almost any disease of the anus, rectum, or sigmoid may be correctly diagnosed.

The author finds the knee-chest posture most convenient for examination. Much can be learned from inspection previous to examination. The condition of the skin, the presence of pruritus, external hemorrhoids, primary chancre, gonorrheal infection, the presence of a fissure indicated by a sentinel pile, the external opening of a fistula, the presence of an abscess by a swelling, and many other conditions can be readily seen by simple inspection. External palpation will reveal the presence of a fistula or an abscess, the position of the coccyx, or the presence of any painful point around the circumference of the anus.

Digital examination is enlightening, and, when carefully made, should cause but little discomfort while revealing a great deal. The finger should be first passed into the anal canal, and gradually, but gently, swept around the circumference, so that any abnormality can be detected easily. The presence of an inflamed crypt, the internal opening of a fistula, hypertrophied papillæ, the presence of a fissure, in fact, a slight abrasion of the mucocutaneous membrane should be detected by the examining finger. If one is not careful, in examining the inside border of the external sphincter, the internal opening of a fistula may be overlooked.

The posterior commissure should be thoroughly inspected, because here one finds most frequently the internal opening of fistulæ. The levator muscle is next inspected, and any abnormality noted, as hypertrophy of this muscle is not infrequently the cause of constipation. The rectum is explored in the same manner as the anus and the presence of a tumor, particularly cancer, can be diagnosed.

As most strictures are within reach of the finger, any narrowing of the bowel can be detected; in fact, any abnormality of the rectum within reach of the finger will be felt.

Proctoscopic examination is perhaps the most helpful method we have for exploration of the bowel from the apex of the sigmoid to the anus. Provided the instrument can be introduced—and it can in the majority of cases—there is no reason why any one familiar with the instrument should not be able to detect any deviation from the normal. Proctoscopic examination is painless when properly handled. One should experience no difficulty except at the juncture of the rectum and sigmoid. It is here that one is most apt to encounter trouble, because the rectum joins the sigmoid at rather an acute angle and this junction is covered by a valve. This difficulty is overcome by gently manipulating the instrument first to the right and then to the left, when the opening will usually come into view. But under no circumstances should any attempt be made to pass the instrument until this opening can be seen. Time and again patients have been referred to the author who were supposed to have a stricture where none existed; and he has not infrequently observed in such cases injury to the bowel, particularly on the posterior wall in the hollow of the sacrum, probably caused by rough instrumentation in an effort to pass the proctoscope from the rectum into the sigmoid. If the inflator, which is a part of every instrument, had been used, less difficulty would have been experienced in passing the tube.

Spasm of the bowel is frequently seen at the rectosigmoidal juncture, and this is sometimes mistaken for stricture; but by waiting a little while, the bowel will be seen to relax, and the instrument can then be passed. The difficulty sometimes experienced in differentiating between diverticulitis and cancer can be cleared up by a proctoscopic examination.

The author has observed little jets of pus shoot forth from diverticuli thus confirming a diagnosis which might have been difficult under any other form of examination, even including the x-ray; because there are certain types of diverticulitis where the bismuth can not penetrate the pockets and the x-ray picture shows only a narrowing of the bowel.

Pigmentation, with its train of symptoms, often suggesting stomach instead of colonic involvement, can readily be diagnosed by using the proctoscope. The various infections, specific and nonspecific, can be differentiated easily under this method of examination. So that all one requires is a knowledge of the normal appearance of the bowel and facility in the use of the instrument.

HEMORRHOIDS

Hemorrhoids are vascular tumors, resulting from dilatation of the blood pools with the formation of connective tissue and tumefaction.

Etiology.—Erect posture, congestion of the liver, constipation, intussusception, or any cause that results in an obstruction to the return circulation may produce hemorrhoids.

Symptomatology and Clinical History.—Hemorrhoids may exist for years and bleed occasionally without causing anything more than a heavy feeling in the anus with more or less obtunded sensation. Patients have stated that they can't tell when they have had a sufficient movement or not. Sometimes they feel they are having a large movement, but find they have passed very little and *vice versa.* After the hemorrhoids are well developed, they come down at stool, but can be readily replaced. Later on, they come down during exercise and do not remain up when replaced. As a result there is loss of sphincter tone, enlargement of the tumors, and general discomfort on walking. On account of the constant moisture, the surrounding skin becomes chafed, adding to the discomfort already existing. Even then some will put up with this inconvenience rather than submit to treatment until a fissure develops, when the intense agony compels them to go to a physician for relief.

Diagnosis.—Proctoscopic examination is the only sure method of diagnosis; because, even though the presence of hemorrhoids is apparent, they are not infrequently associated with cancer or other serious ailments. Moreover, bleeding at stool may be due to a fissure, an infection of the bowel, a polypus or various other diseases, so that it is always best to rule out by a proctoscopic examination all possible complications before classifying the case as hemorrhoids pure and simple.

Complications and Sequelæ.—Abscess is probably the most frequent complication, and a fistula may follow an abscess. Occasionally, gangrene and sloughing, as the result of prolapse and edema, may be followed by fatal infection of the portal circulation;[*] or stricture may be the only consequence. Loss of control is always a serious problem, especially when it attacks the old. Many people are treated at heart and lung clinics for shortness of breath without suspecting a secondary anemia due to gradual loss of blood from an ulcerated hemorrhoid. The author has seen many patients with a hemoglobin of 35 per cent. restored to health shortly after having hemorrhoids removed.

Association with Other Diseases.—The diseases with which hemorrhoids are most frequently associated are *cancer,* stricture, intussusception, cirrhosis of the liver, abdominal tumors, and diseases of the uterus.

Clinical Varieties.—Hemorrhoids are divided clinically into two classes, external and internal. The external hemorrhoids are further subdivided into varicose external hemorrhoids, which are merely engorgement of the veins surrounding the anus; thrombotic external hemorrhoids, which are oblong, pear-shaped tumors, usually resulting from thrombosis or hemorrhage beneath the skin, and are ushered in with a severe, sharp pain, followed by tumefaction and spasm of the muscle; and skin-tabs which are projections of the skin, associated with pruritus, thrombotic hemorrhoids, syphilis, or gonorrhea.

Internal hemorrhoids are divided by some into venous, columnar, and nevoid. The latter is not in reality a hemorrhoid, but a vascular tumor usually of congenital origin.

Treatment.—The treatment of hemorrhoids is either palliative or operative. Very frequently all that is necessary is to **regulate the patient's bowels** and apply some **astringent ointment.** Where the hemorrhoids have prolapsed and there is considerable edema, the continuous application of **gauze wrung out of hot water** will relieve a great deal of suffering. The following ointments have been found useful:

℞
Unguenti acidi tannici..................... ℥i (31.10 grams)
or
Unguenti acidi tannici..................... ʒiv (15.50 grams)
Unguenti stramonii .... ʒiii (11.65 grams)
Unguenti belladonnæ .... ʒi (3.90 grams)
M. Sig.—Apply locally three or four times a day.

Injection into the hemorrhoid of a **5 per cent. solution of quinin and urea hydrochlorid solution** sometimes gives immediate relief for a year or two. It can be repeated when found necessary.

PAPILLITIS AND CRYPTITIS

Etiology.—At the internal sphincter there are a number of semilunar depressions; and between each semilunar depression a column. These columns contain an artery and a vein, and are well adapted for the protection of blood-vessels. There are also a number of sensory bodies known as papillæ, which contain a rectile tissue and occasionally undergo a very marked hypertrophy. As the result of this hypertrophy these bodies occasionally protrude outside of the anus during defecation and are not infrequently mistaken for adenomata. Unlike adenomata, they do not bleed, but are often responsible for pruritus ani and spasm of the sphincter. When they reach this size they should be removed. Inflammation of the crypts is a much more serious condition, as infection, abscess and fistula may result.

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[*] Such a case is reported by Mummery and Toshi, where a fatal result occurred following gangrene of the hemorrhoids (Death from Strangulated Internal Hemorrhoids, *Lancet,* London, Feb. 13, 1915, i, 322).

Symptomatology.—The most prominent symptom is a sharp, lancinating pain, occurring during defecation, which, later on, may become dull and throbbing. On account of the invisibility of the ulcer, the pain may be mistaken for a neuralgic condition. Occasionally in the male the pain is referred to the prostate and occasions painful urination. Spasm of the sphincter, constipation, and abdominal discomfort may form part of the picture.

Diagnosis.—This can be made only by gently probing the valves, when, if any lesion exists, a pain somewhat similar to that caused by an exposed nerve in a tooth, will be experienced.

Treatment.—The treatment is **medical** and **surgical**. The medical treatment consists in making **applications of pure ichthyol**. This can best be done by using a bent probe passed through the analscope and then into the base of the valve.

FISSURE

Etiology.—Fissure in ano is caused by a solution of continuity of the mucocutaneous membrane, due to the passage of hard fecal matter or foreign bodies.

Symptomatology.—There is always some distress after a movement of the bowel in this condition. It is usually a burning sensation which lasts anywhere from a few minutes to several hours, or possibly till the next movement when it increases in intensity. Associated with the movement may be a few drops of blood, or the fecal matter may be merely streaked with blood.

Fissure is frequently associated with hemorrhoids, but the latter are not discovered until the fissure occurs. This has led to the belief that hemorrhoids are always painful, but it is usually pain from the fissure that the patient feels. There is more discomfort than pain associated with hemorrhoids, as a rule.

Diagnosis.—On examination in the knee-chest posture or on the side, you find an elliptical ulcer with overhanging edges. The floor of such an ulcer is bright red in color, and covered with serum or blood. The instrumental examination is usually so painful that it should be avoided if possible. It is perfectly easy to see the lesion by gently separating the parts, asking the patient at the same time to bear down.

Complications.—The complications arising from this lesion are numerous and far-reaching. The most noticeable are difficult and painful urination, sciatica, constipation, indigestion, visual disturbances; in fact, pronounced sympathetic upset.

Sequelæ.—The sequelæ are abscess, fistula, loss of weight, neuroses, mental disturbances; in fact, the effects are often far-reaching.

Treatment.—Treatment is **medical** and **surgical**. Surgical treatment consists in **division of the sphincter**. The author does not approve of divulsion. If taken very early, medical treatment in a large percentage of cases results in cure. This usually consists in the application of a **one per cent. nitrate of silver, pure ichthyol**, painting with **chloral hydrate**, or the use of the following prescription:

℞

Ichthyol	ʒi	(3.90 grams)
Argyrol	ʒii	(7.80 grams)
Anesthesini	ʒss.	(1.95 grams)
Petrolatum	q.s. ad ʒi	(31.10 grams)

M. Ft. Ung.
Sig.—Apply locally.

ABSCESSES

An abscess is a collection of pus following the invasion of the tissues by some pathogenic microörganism. Abscesses form about 25 per cent. of the cases seen in proctologic practice. They are found both above and below the pelvic diaphragm. The perirectal, those occurring around the margin of the anus, are the ones most frequently seen, and are due to some abrasion of the skin or infection of a hair follicle or sebaceous gland. When seen early and opened, they do little damage, but if allowed to go on, they follow the blood-vessels and open into the anus.

ABSCESSES OF PERIANAL REGION

Abscesses of the gland of Bartholin not infrequently extend to the rectum. It is extremely important for this reason that they should receive early attention.

Abscesses that occur underneath the perianal fascia may be due to superficial abscesses or result from fissure. When occurring anteriorly they may extend to the scrotum, resulting in an enormous collection of pus in this loose tissue.

Symptomatology.—They may be ushered in by a chill with slight rise of temperature, or merely by a feeling of discomfort, followed by pain, whether the patient be sitting, standing or walking. They are aggravated by a movement of the bowels. If not opened, swelling and tenderness result.

Treatment.—The treatment consists of **crucial incision** followed by **irrigation and sitz-baths.**

ABSCESSES IN POSTERIOR TRIANGULAR SPACE

Abscesses in the posterior triangular space, which is an imaginary triangle with its base between the two sphincters posteriorly and its apex at the tip of the coccyx, are very important, in that nearly all ischiorectal abscesses are the result of infection of this space. They usually result from a fissure in the posterior commissure. Fortunately not all fissures are followed by infection; nor does an ischiorectal abscess always follow an infection of the triangular space. However, all fissures may be looked upon as potential abscesses; and likewise, abscesses of the triangular space may be considered potential ischiorectal abscesses. For this reason the importance of early treatment will be readily appreciated.

Because of a depression between the two sphincters posteriorly, drainage is imperfect; and when there is a lesion in this space, pus collects and bacterial invasion of the deeper tissues is augmented.

It is surprising how frequently abscesses in this triangular space run a chronic course. This may be due to the fissure, which usually precedes abscesses, causing formation of connective tissue which walls off the abscesses when formed. It is noticeable that in most instances there is a large opening surrounded by a ring of connective tissue.

Diagnosis.—The diagnosis is usually made from a preceding history of fissure and swelling at the site of the abscess and by a digital exploration. The history is that of pain during and following a movement of the bowels. There are then noticed a swelling, great discomfort, difficulty in urination, severe pain on standing or sitting. This lasts for three or four days when there is discharge from the anus, followed by relief.

Treatment.—The treatment is **posterior incision** which is **carried up laterally on both sides,** for about half an inch, followed by **frequent irrigation.**

ISCHIORECTAL ABSCESSES

Ischiorectal abscesses are the result of traumatism, infection of the skin, fissure in the posterior commissure, ulcerated hemorrhoids, cryptitis, and perforation of the anus by foreign bodies. It is surprising how frequently this abscess occurs in tuberculous people. The offending organism is quite often the bacillus of tuberculosis.

Symptomatology.—The constitutional symptoms are usually pronounced; slight chills, temperature and headache, associated with severe pain during defecation.

Diagnosis.—The buttocks are red, swollen and shiny. Fluctuation occurs over the center of the abscess, and there is difficulty in urination.

Treatment.—When the diagnosis has been made, the abscess should be **opened by a longitudinal incision,** and the **trabeculæ** which cause pocketing of pus should be **broken down.** The subsequent treatment consists in **frequent irrigations** with **peroxid of hydrogen,** 1 per cent., or **permanganate of potash,** 1 to 10,000.

SUPERIOR PELVIRECTAL ABSCESSES

Abscesses occurring above the pelvic diaphragm are much more serious than any we have mentioned as they are much more likely to result

in serious damage to the bowel if improperly handled and may be followed by general sepsis.

Etiology.—Superior pelvirectal abscesses usually result from ulceration of the anterior wall of the rectum and from prostatic or seminal vesical infection. Other causes are tubal or peri-uterine infections. It has resulted sometimes from the Whitehead operation or partial excision of the rectum.

Symptomatology.—The symptoms consist of a feeling of weight in the rectum, and of a desire to defecate. These may be accompanied by chills, fever, and headache.

Diagnosis.—The index finger is introduced into the rectum. A soft, boggy mass will be felt anteriorly.

Complications.—Posterior pelvirectal abscess, abscess opening into the groin or into the rectum (resulting in a urethral-rectal fistula) may complicate superior pelvirectal abscess. Posterior pelvirectal abscess may follow a superior pelvirectal abscess, and has usually the same etiology. It can be diagnosed in the same manner, except that the abscess is in the posterior instead of the superior wall of the bowel.

Treatment.—This is entirely surgical.

FISTULA

Definition.—A fistula is a canal resulting from an imperfectly drained abscess. This canal may have an opening in the skin or in the mucous membrane of the rectum, or it may communicate with the urethra (recto-urethral fistula), or with the vagina (rectovaginal fistula). The internal openings are usually few; the external openings may be single or multiple. The author has counted as many as fifty external openings in one case. Fistulas are spoken of as external, internal, complete, superficial, or deep, depending upon the origin of the infection.

Etiology.—The etiology of all fistulas can be summed up in a few words; infection, abscess, discharge, fistula.

About 5 per cent. of all fistulas are of tuberculous origin. It does not necessarily follow that because a patient has pulmonary tuberculosis that the fistula is always tuberculous. Some of the most difficult fistulas to handle are those that have their origin in the hip joint, and in the coccyx. Those associated with osteoperiostitis of the coccyx, or fistulas causing an infection of the coccyx, are very intractable, unless the coccyx is removed.

Symptomatology.—If the fistula drains well, there is no particular discomfort, except that associated with the discharge; but in many instances there is pain during defecation; also pain during any form of exercise. The discharge from a fistula ceases in time, and the patient considers himself cured until he is surprised by another swelling which ruptures not at the old opening, but at some point distant from the first.

Diagnosis.—The diagnosis is usually made by inspection and digital examination. It is seldom necessary to cause the patient severe pain by probing a sinus, as there is sufficient outward evidence to establish the presence of fistula. Besides, nothing can be gained once the presence of a sinus is established by probing, as all necessary information can be obtained at the time of operation with the patient under an anesthetic. The location of the external opening, whether it is anterior or posterior, furnishes considerable information.

Pain on sitting, rising, walking upstairs, or other exercise, together with the discharge from the anus, should always suggest the possibility of fistula.

If the anterior opening is within an inch and a half of the anus and anterior to a line drawn from one tuberosity to another bisecting the anus, the internal opening will usually be found anteriorly just inside of the external sphincter. If the external opening is posterior to this line and the same distance from the anus, the internal opening is usually found in the posterior commissure between the two sphincters.

Treatment.—This is **surgical**. All fistulas are curable if properly treated; and this includes the tuberculous variety. If the patient is not within reach of a surgeon, keeping the sinus well drained will materially add to his comfort and prevent further complications. If a fistula is seen early and properly cared for, the serious complications that may follow as the result of neglect may be prevented.

PRURITUS ANI

Definition.—Pruritus is an itching around the anus, following infection of the skin. It may be initiated by constipation, hemorrhoids, fistula, fissure, papillitis or cryptitis, and is commonly aggravated by scratching. There are few conditions that have such a far-reaching effect in undermining the constitution. Once it has started it may continue for a long time, even though the original cause has been removed.

Etiology.—Murray held that pruritus is always due to an infection of the skin by the streptococcus. This is true to a limited extent. The author has been able to verify this in only a few cases. In all the cases which the author has seen, he noticed that the following condition is always present—a somewhat pronounced congestion of the anus associated with rectal constipation.

It is very difficult to trace the origin of this trouble to any one of the innumerable causes that have been described, except papillitis; if this exists, the patient will state that his itching began with a feeling as though worms were being passed.

Diagnosis.—The diagnosis is usually made by the patient, who has inherited the impression that itching is always associated with piles; so that he invariably tells you that he is suffering from "itching piles." How the two conditions came to be associated, the author does not know, except that piles are occasionally present with pruritus. On inspection, the skin will show evidence of scratching. It is thrown into folds and may be macerated and moist or dry and shiny. There may also be a number of skin tabs surrounding the anus. The disease may be limited, or very extensive, spreading to the buttocks, to the scrotum in the male, or to the labiæ in the female. In some persons it involves the mucocutaneous surface. In all cases, it is much worse at night than during the day. It prevents sleep, and in many results in loss of weight with pronounced manifestations.

Treatment.—The treatment is as varied as the causes of the disease. As far as the author's experience goes, there is no remedy that is specific for all cases. There are, however, a few general rules that are applicable in all instances, namely, keeping the **parts thoroughly cleansed with alcohol or witch hazel**; keeping the **bowels open**; and instructing the patient to **wash out the anus** with an ounce or two of water or a 10 per cent. solution of witch hazel **after each movement.** When the skin is macerated and there is quite a bit of moisture, ointments should never be used. In such instances, **calamine lotion** will be found helpful.

Painting all abrasions with **nitrate of silver,** 2 per cent., is advised. Other remedies that have merit are **stearate of zinc,** dusted on the parts, **plain starch** and **zinc oxid.** Painting with **tincture of iodin,** and then using powdered starch to remove the excess of iodin, has proved beneficial. A 2 per cent. **solution of carbolic acid** is also soothing; or the following prescriptions may be used:

℞

Acidi carbolici	ʒii	(7.8 grams)
Acidi salicylici	ʒi	(3.9 grams)
Glycerini	℥i	(31.10 grams)

M. Sig.—Apply with camel's hair brush when itching is severe.

or

℞

Calaminæ	Grains xl	(2.6 grams)
Zinci oxidi	ʒiii	(11.65 grams)
Glycerini	℥i	(15.5 grams)
Liquor carbonis detergens	♏xx	(1.44 gram)
Aquæ calci	℥i	(31.10 grams)
Aquæ	q. s. ad. ℥iv	(124.4 grams)

M. Sig.—Apply twice daily.

It is needless to say that all cases of pruritus should be given thorough local and general examination. If possible, find the cause and remove it.

Autogenous or **stock vaccines** of the **streptococcus fæcalis** have, in a few instances, given great relief.

An **anticonstipation diet** is always indicated. Inasmuch as in some instances such foods as shell fish, strawberries, eggs, and coffee have been known to aggravate the disease, they should be given consideration when prescribing a diet.

Many patients have stated that they were entirely free from itching when taking **aspirin.**

DISEASES OF THE COCCYX

As the result of injury or infection, pain in the coccyx may become so severe as to partially or completely incapacitate the patient. The author has observed as a result of the fistulous or postanal dimple severe osteoperiostitis of the coccyx; and, while not occurring with any frequency, still one should be constantly on the lookout not to overlook this complication. So-called coccygodynia, which is a neuralgia of the sacrococcygeal joint resulting from injury, is not as continual or as painful as osteoperiostitis, but, at times, it is quite intractable.

Treatment.—In osteoperiostitis **removal of the coccyx** is always followed by gratifying results. In the neuralgic conditions **salicylates, rectal injections of warm, alkaline solutions,** or **small injections of alcohol, hypodermatically,** give relief; however, it is also sometimes necessary to **excise the coccyx.** This operation is easily performed under local anesthesia.

PIGMENTATION OF THE BOWEL

Pigmentation is a coloring of the bowel, varying between an ocher and a muddy brown shade. It is probably due to a deposit of pigment as the result of bacterial action, and a train of symptoms are associated with it, which might lead one to suspect that the trouble was in the stomach rather than in the rectum and colon.

Some ten years ago, the author called the attention of the profession to this unusual condition; and later, McFarland, after working with him treated the subject in great detail.

Symptomatology.—The symptoms are very marked toxemia, hyperacidity of the stomach, eructations after eating, bloating, periodical attacks of headache, and pronounced constipation. The odor of the stools is noticeable, having a sour pungent smell. Also there may occasionally be bloody and mucous discharges from the bowel.

Diagnosis.—This can be made only by proctoscopic examination, at which time the pigmentation is demonstrated.

Treatment.—The treatment is largely **dietetic** and the author is in the habit of **eliminating proteins** as far as possible. A **vegetable diet** gives the best results. The hyperacidity of the stomach can be relieved by **peroxid of hydrogen,** a teaspoonful in a wine glass of water. The bowels should be kept open, and the following prescription has been found helpful:

Extracti rhamni purshianæ (Cascara sagrada)...........ʒii (7.8 grams)
Extracti belladonnæ foliæ.........................grains ii (.13 gram)
M.—Divide into 30 capsules.
Sig.—One capsule at bedtime.

The colon should be irrigated once a day with a 2 per cent. solution of peroxid of hydrogen or **permanganate of potash.**

Prognosis.—It takes from six months to a year and a half, occasionally longer, to effect a cure; but the prognosis, as far as the author has seen (and he has had a number of cases), is favorable.

DIVERTICULITIS OR DIVERTICULOSIS

Diverticuli are flask-shaped protrusions, sometimes occurring singly, but they are oftener multiple, and may be in rows or clusters. They may appear anywhere in the alimentary canal. Most frequently, however, they are confined to the sigmoid colon. They are potential troublemakers in that they may at any time give rise to a series of pathological processes and clinical symptoms resembling appendicitis.

Etiology.—The cause of diverticuli has been the subject of much speculation; but most of us now agree that any cause that brings about a weakness of the muscular tone may result in hernial protrusion of the mucosa. Fat people seem to be more prone to this visitation; and constipation with the accumulation of fecal material, and distention of the bowel with gas are undoubtedly contributing factors. Naturally, they are most apt to occur in the elderly, but occasionally attack the young. As they are usually found in the appendices epiploicæ, Bland-Sutton has suggested that the fat of the submucous coat is directly continuous with that of the appendices epiploicæ, and so exerts a predisposing influence on these hernial sacs. One of the most interesting specimens of diverticulitis which the author has seen occurred in a comparatively thin woman upon whom he operated for intestinal obstruction. In this instance, one small diverticulum of the hundreds present became attached to a loop of small bowel and caused obstruction. As far as he could observe there was no evidence of pathology in any of the other diverticuli.

Symptomatology.—The symptomatology varies with the degree and type of inflammation. The usual history is that of chronic indigestion associated with a dull, aching pain in the left side, but in some instances merely with discomfort. Constipation, the passage of blood-stained mucus and gaseous distention, together with acute exacerbations of temperature with a marked general toxemia should suggest diverticulitis.

In one case which the author saw, the patient complained of constipation, headaches, toxemia, with occasional temperature; and the first intimation he had of anything serious was the passage of gas and feces from the penis. Another patient, a woman, gave a history of failing health, indigestion, and the occasional passage of mucus and blood extending over a period of years. It was not until a proctoscopic examination had been made that the author realized that he was dealing with a case of diverticulitis.

Sometimes the disease is ushered in with acute abdominal pain; at first, it is referred to the navel, or to the pit of the stomach, gradually locates itself in the left iliac fossa, and is associated with nausea and vomiting, abdominal rigidity and tenderness. The fulminating type may begin with severe abdominal pain, rupture of the abscess, and general signs of peritonitis.

Diagnosis.—The diagnosis is usually made from the symptoms mentioned above, the proctoscopic examination, and the presence of a tumor located on the left side. Of course, in acute cases with sudden onset, there is not always time to study the case exhaustively and one must be satisfied with the diagnosis of acute abdomen. In the chronic cases the history of pain, constipation, and the passage of mucus and blood should lead to an early examination. In chronic cases, a proctoscopic examination gives about as much information as any other method.

DIFFERENTIAL DIAGNOSIS.—It is sometimes quite difficult to differentiate diverticulitis from various abdominal troubles which may simulate it, especially an acute appendix with left-sided symptoms. One patient well illustrated this situation. All the symptoms were left-sided, and, on opening the abdomen, an inflamed appendix was found adherent to the sigmoid. A rectal examination, in most instances, will settle the question, by palpation of the tumor on the right or left side. Other diseases from which diverticulitis must be differentiated are pelvic inflammation, an inflamed and strangulated ovary, and acute nonspecific inflammation with colitis and pericolitis. The specific inflammation and cancer can be differentiated by a proctoscopic or pelvic examination.

Treatment.—The treatment is **always surgical**, and varies with the clinical type presented. The author feels that a stoma above the inflammation will prevent reinfection and make it much easier to perform a resection later on. The very acute conditions demand, of course, immediate surgical relief.

Pathology.—There may be: general infection of the peritoneal cavity from thinning of the sac wall; gangrenous inflammation; chronic, hypertrophic, or peridiverticulitis; adhesions between the diverticulum and the small intestine of the bladder; local abscesses, with subsequent burrowing of a fistula in the wall of the bowel; chronic inflammation of the sigmoid mesentery; and carcinomatous degeneration.

INFECTIONS—SPECIFIC AND NONSPECIFIC

The division of infections into specific and nonspecific is the most convenient for clinical study. Under the heading of specific infections there is included all of the ulcerative and infectious processes which cannot be ascribed to any definite organism. There is some reason to believe, with but little proof to substantiate it, that some member of the streptococci family will be eventually held responsible for this form of infection.

Specific infections are amebic, diphtheritic, syphilitic, gonorrheal, and tuberculous rectitis.

So much has already been written about amebic rectitis that only the salient, diagnostic points shall be mentioned. An impression prevails that dysenteric proctitis is a disease of tropical climates. This is far from true, as the author is constantly seeing patients suffering from amebic infection who have never been away from the vicinity of New York. It is also erroneously believed that diarrhea is inevitably a constant accompaniment of dysentery. The fact is that there are many instances in which patients with constipation alternating with diarrhea suffer with amebic infection. A dose of epsom salts, and a warm stage of the microscope with some mucus from the stool will readily disprove that diarrhea always exists in amebic dysentery.

The picture as seen through the proctoscope is fairly characteristic. On passing the instrument into the rectum, we see a number of scattered papules with a little vesicle on top of each, or a necrotic spot; or, maybe, an ulcer with a narrow opening on top; and the mucous membrane covered with a serous exudate mixed with mucus.

If further proof is needed, the organisms, if present, may be demonstrated by examining upon a warm stage of the microscope some of the exudate which has been obtained by passing a platinum wire through the proctoscope to the site of the lesion.

Syphilis of the Rectum

To syphilis has been attributed a great many pathological processes of the rectum; of this there is hardly sufficient proof. For instance, syphilis is presumed to be the cause of the large percentage of strictures, but the author has seen very few that he believed to be of syphilitic origin. Of primary chancre of the anus, the author has observed 6 cases in twenty years. The apparent low percentage of the disease in New York would indicate that it is a rare condition; but the truth is that its lack of symptoms and close resemblance to fissure undoubtedly account for its not being diagnosed more frequently. All the cases which the author has seen were referred to him with a diagnosis of fissure.

Symptomatology.—Soreness, and pain occasionally, during defecation are the only symptoms.

Diagnosis.—The diagnosis should be easily made. The presence of a fan-shaped extension of the mucous membrane, slightly raised above the skin, bright red, when seen early, dusky red, in later stages, should denote the presence of a chancre. In none of the author's own cases was there any noticeable induration; and in each one the diagnosis was confirmed not alone by a Wassermann but by the recovery, previously, of the spirochetæ from the inguinal glands.

Condylomata

Other evidences of syphilis observed in the region of the anus are the condyloma lata. They are of a pearly gray color with a fetid odor and acrid discharge. They should be distinguished from the condylomata acuminata, which are due to an inflammation of the papillary layer of the skin, and usually associated with gonorrhea. They are of a pinkish appearance and not flattened as are the condylomata lata.

Gumma

There are innumerable cases reported; but the author has seen but few cases in twenty years. It would seem probable that the reason the early literature is so voluminous is that there was then no confirmatory method of positive diagnosis. The author is of the opinion that in most instances in which gummata were reported they were dealing with a nonspecific, follicular, nodular inflammation, rather than a syphilitic one. It does not always follow that because a patient has syphilis that a nodular rectitis is syphilitic; as in so-called syphilitic stricture the only proof positive would be the finding of the spirochetæ in the tissues. The symptoms of syphilitic inflammation in the rectum differ in no wise from those of an ordinary inflammation. Fournier, in his book on "Tertiary Lesions of the Anus," speaks of an infiltrating rectitis, involving the submucous layer without ulceration of the mucosa, and comparable to the cirrhotic changes which take place in the spinal cord and other organs. It is quite possible this is so, but one doesn't see such conditions nowadays, and this may be due to the fact that syphilis is treated more vigorously and at an earlier stage than formerly.

Treatment.—The treatment is **antisyphilitic.**

GONORRHEAL RECTITIS

Gonorrheal rectitis is a fairly common occurrence; but the author cannot agree with those who state that it involves not alone the rectum but the colon, and may result in stricture. The cases that have come under his observation, and they are numerous, have all been limited to the first three or four inches of the rectum. The mucous membrane is dark red in appearance, edematous and swollen, and is attended by pain, pressure and a desire to move the bowels. The anus is contracted, congested, and the opening is apparently very small. If ulcerations occur, other bacteria may take part in the process, and serious inflammation follow involving the submucous layer. However, this is fortunately rare. As the disease tends to run its course within a few weeks, few complications occur.

Treatment.—This consists in **irrigations of warm boric solution** followed by an **injection of 5 per cent. argyrol.**

CHANCROIDS

Chancroidal ulceration, while frequently seen around the anus, is rarely seen within the rectum. It is much more common in females than in males, and this is accounted for by the vaginal discharges trickling down over the perineum. According to Pean and Malassez, one-half of the superficial anal ulcerations seen in females were due to chancroids. These are usually found between the folds of the skin, extending into the mucocutaneous surface; they are commonly multiple and sharply defined; the skin is macerated; and the ulcerations are painful when the sphincter is involved.

Treatment.—The treatment is **cleanliness** and **irrigations** with **permanganate of potash,** 1 to 10,000.

TUBERCULOUS

Tuberculous ulcerations of the anus usually follow an abscess. The appearance is that of an irregular, undermined ulcer, with an unhealthy base with worm-eaten appearance. Liberal application of the **cautery,** followed by **daily irrigations** of **permanganate of potash,** 1 to 10,000 will in most instances result in a cure.

These ulcers may extend to the rectum, when the problem of dealing with them becomes much more serious; but even then one should not give an unfavorable prognosis, because the same line of treatment, if vigorously pursued, even though it must be continued for a year or longer, as has been necessary in some cases, in the end will no doubt be gratifying.

But when, as a result of intestinal tuberculosis, the rectum becomes secondarily involved, there is no treatment known that can effect a cure. Primary tuberculosis of the bowel is rare. In cases secondary to pulmonary tuberculosis the outlook is hopeless. There is, however, a form of tuberculosis, namely the hyperplastic type, in which there is a proliferation rather than a destruction of tissue. This condition usually involves the upper rectum or sigmoid, and is amenable to **surgical intervention.**

NONSPECIFIC INFECTION

There are all gradations of this infection, from a slight, superficial inflammation of the mucosa to an acute and chronic infection, involving all coats of the bowel from the rectum to the stomach. It may be discontinuous and patchy, or continuous with a granular exudate resembling fish spawn covering the entire mucosa. The mucosa membrane may be bosselated or entirely destroyed.

Symptomatology.—The symptoms vary with the pathology. The disease may come on suddenly, with diarrhea, blood, mucus and pus. The temperature may be high 104° F. (40° C.), resembling a case of typhoid in the third week. Or there may be merely gastro-intestinal symptoms with passage of small quantities of blood. In some cases there is constipation with intermittent attacks of diarrhea and marked gastric symptoms with high acidity. Diarrhea may be continuous with the passage of large quantities of pus, due to the formation of intramural abscesses. The stools are usually very fetid, sometimes overpowering.

Diagnosis.—This is usually made by an examination of the stool and by proctoscopy. To those not familiar with the disease, all specific organisms must be ruled out by microscopic examination.

Sequelæ.—Endocarditis, occasionally, is associated with nonspecific infections. In a small percentage perforation occurs. In 3 cases the bowel ruptured spontaneously and was followed by peritonitis and death. Stricture or strictures usually follow in patients with a subacute infection if not properly treated. In one of the author's patients, when first seen, the stricture had extended from the hepatic flexure to the anus; in another, from the hepatic flexure to the sigmoid.

Treatment.—All cases should be given medical treatment for three or four months. This should consist of **rectal irrigation twice daily.** The solutions found most useful are **permanganate of potash,** 1 to 10,000; and **hydrogen peroxid,** 1 per cent. The **Carrel-Dakin solution** is sometimes used. If at the end of four months the condition is not greatly improved, a **stoma** should be made, oral to the infection.

Prognosis.—The prognosis depends upon the degree and extent of the inflammation. The majority of cases, however, get well within a year and a half, if treated patiently and systematically.

Pathology.—The pathology is that of an acute and chronic inflammation.

STRICTURE

A stricture may be defined as a diminution in the caliber of the bowel, as a result of either an encroachment by a tumor, or by cicatricial contraction following inflammation.

Etiology.—Most strictures are due to a low-grade inflammation causing an increased production of connective tissue between the muscular bundles and a consequent diminution of blood supply. This is well illustrated in resection of the rectum for carcinoma. There are, however, strictures found in infants due to a septum. As a general rule such malformations are easily corrected, as the bowel above the septum is commonly found to be healthy. Some writers mention stricture as the result of a spasm of the intestine; and in such instances the contraction is no doubt the result of muscular hypertrophy.

Symptomatology.—The symptoms are gradually increasing constipation, with the passage of blood, mucus, and pus, and a frequent desire to move the bowels, which, at times, is imperative.

The impression seems to prevail that tape-like stools are an indication of stricture; but unless the stricture is close to the anus this is not so, because the fecal matter always takes the form of the final opening through which it passes. This form is more often indicative of spasm of the sphincter than of stricture.

Diagnosis.—The diagnosis can be made by digital examination, as most strictures of the rectum are within reach of the finger. When situated higher up, as in the sigmoid, a proctoscopic examination becomes necessary.

Complications.—These are endocarditis, nephritis, and intestinal obstruction.

Clinical Varieties.—These are congenital, spasmodic, inflammatory and neoplastic.

Treatment.—Strictures of large caliber can be materially improved by **frequent irrigations** with **antiseptic solutions.** But it will usually be necessary to resort to an **artificial anus** in order to achieve the best results.

Prognosis.—It is needless to say that a patient suffering from stricture is seldom wholly cured; but when properly handled a good functional result is obtained.

Pathology.—Simple, inflammatory hyperplasia of the involved structures is present.

PROLAPSE

Prolapse of the bowel may be complete or incomplete.

Incomplete Prolapse

Incomplete prolapse is an invagination of the mucosa.

Etiology.—Straining at stool during infancy, relaxation of the muscle in old people, and polypoid growths are the usual causes of prolapse.

Symptomatology.—Bleeding, with prolapse of the mucous membrane, is the only symptom.

Diagnosis.—The diagnosis is made by having the patient strain, when usually a funnel-shaped piece of mucous membrane appears. However, the mucous membrane may prolapse all around. In such instances it will be distinguished from a complete prolapse by observing that the folds are longitudinal and radiate from the center to the circumference.

Treatment.—Our first effort should be directed toward removing the cause. **Straining at stool** should be **interdicted.** The best plan is to have the patient move his bowels without any effort. To accomplish this an **oil enema** should be given previous to a movement. **Astringent enemas**, such as **krameria**, will be found effective. If the muscle is relaxed and the mucosa prolapses between movements, **strapping the buttocks** may be tried. As a general rule, the cause being removed, and the muscle tone improved, the prolapse disappears; and this is particularly true in infants. However, in older people **surgery** may be necessary; the operation may be performed under local anesthesia.

Complete Prolapse

Complete prolapse is an invagination outside of the sphincter of all the coats of the bowel.

Etiology.—The factors which enter into complete prolapse are long sigmoid, deep culdesac, relaxation of the lateral ligaments, constipation and straining at stool. A full bladder has also been regarded by some as an etiological factor.

Symptomatology.—Gradual protrusion at stool, with occasional bleeding and discharge of mucus is present. As time goes on, if not attended to, the protrusion increases. As a result, the sphincter becomes relaxed and incontinence follows. At first, the prolapse can be replaced but in later stages on coughing or sneezing the prolapse comes down.

Diagnosis.—Ordinarily, when the prolapse is not evident, the diagnosis can be made by asking the patient to strain; or, after giving a peroxid enema the bowel will protrude. However, it may be found necessary to use the proctoscope.

Treatment.—In the early stages the treatment already described in incomplete prolapse may be sufficient. But when the condition is well established some operative procedure becomes necessary. The operation which the author has devised of **resection of the sigmoid and shortening of the lateral ligaments** is anatomically correct and will give a good result.

RUPTURE, INJURIES AND WOUNDS

Rupture, injuries and wounds of the rectum, while comparatively rare, are always serious. On account of the anatomical relationship to the bony wall of the pelvis and its muscular padding on both sides, the rectum allows considerable traumatism without serious injury. But at the rectosigmoidal juncture, injuries more easily happen and more frequently occur than lower down. Hellsstrom, in a review of the literature of spontaneous rupture, is convinced that, in most instances preceding the rupture, there was some pathological condition. He relates the case of a youth, seventeen, previously healthy, who experienced pain and bleeding at defecation, followed by rupture with profuse peritonitis. Another patient of Hellsstrom's, sixty-three years old, was aroused in the night with unbearable pain in his rectum. At operation, ten hours later, a rupture with smooth edges and no evidence of blood in or around it, was discovered. There was no history of injury.

During the late war, there were a great many cases of projectile wounds of the rectum reported. A case reported by Heincke, in which a healthy man, thirty years old, while lifting a heavy piece of iron experienced great pain and vomiting. When admitted to the hospital, the rectum was found to be ruptured. The author has treated several cases of severe lacerations of the perineum and rectum which were caused by a fall on some sharp body. In all instances the shock was pronounced.

Symptomatology.—Severe pain in the rectum followed by shock, with or without hemorrhage would suggest injury in this region.

Diagnosis.—The diagnosis is usually made by inspection or proctoscopic examination.

Treatment.—This is **surgical.**

Prognosis.—This should be guarded.

FOREIGN BODIES

Some quite extraordinary foreign bodies have been recorded as found in the anus. In the majority of these instances they are taken into the stomach, passed through the bowel and become implanted in the rectum. Pierra reports a case in which a stick 9½ inches long and inches in diameter was recovered from the rectum without serious damage.

The South African kaffirs have been known to swallow diamonds, recovering them later on. Some of the natives of Bengal were accustomed to putting a plug of dried clay in the rectum after the bowels moved, allowing it to remain there until the next movement. Fishbones and needles, however, are the objects most commonly found in the rectum.

Symptomatology.—The symptoms will depend upon the kind of foreign body and the location. An abscess may be the first indication. Usually, however, as in the case of small objects, a sharp, shooting or stabbing pain during defecation is experienced. In one patient of the author the foreign body perforated the bowel and lodged in the sacroiliac joint.

Diagnosis.—This is usually made by the examining finger, the proctoscope or the x-ray.

Complications and Sequelæ.—Abscess formation and fistula, suppuration between the rectum and other organs are the most serious complications.

Treatment.—This consists in **removing the foreign body** under an anesthetic. It may be possible, if the objects are not too large, to remove them through the proctoscope.

TUMORS

A benign tumor is a new growth whose cells are arranged in the same order as the tissues from which it is derived, with no special function, and with a tendency to grow away from the parent tissue. The benign tumors usually found in the rectum are adenoma, papilloma, myoma, fibroma, villous growths, fibromyoma, angioma, chondroma, lipoma, lymphadenoma, cystoma, and dermoid cysts. As it will be impossible, in a limited space, to treat each of these tumors separately, only those most frequently seen will be considered.

Benign Tumors

The *adenomata,* a glandular type, may be single or multiple; they may have a pedicle or a broad base. When occurring singly, as they do in young children, they ordinarily have a long pedicle. When multiple, they are always associated with inflammation of the bowel and nearly always degenerate into adenocarcinoma.

The symptoms are the passage of blood and mucus, occasionally severe diarrhea and tenesmus.

Villous growths are broad-based tumors with a number of villous tentacles.

The symptoms are pain in the back with bloody diarrhea and tenesmus. This type of tumor nearly always undergoes carcinomatous degeneration and should be removed early.

The treatment of all benign growths is **surgical.**

MALIGNANT TUMORS

Malignant tumors are carcinomatous and sarcomatous. In the 700 cases seen by the author 1½ per cent. were sarcomatous. Nearly all the malignant tumors found in the rectum and sigmoid are of the adenocarcinomatous type.

Etiology.—Stockard in his studies of normal growth has devoted some attention to abnormal growths as well, particularly teratoma. He has found that the rules which govern normal growth also have a special bearing on the development of cancer. He is convinced that there is a common condition of balance between growing parts and the potential growth parts; and all parts of the body have what he terms the advantage of position until they attain their full growth; that, if for any reason any part loses this advantage before having attained its full growth, it becomes dwarfed and some other organ assumes the advantage; that regulated balance among the organs is to a great extent due to the inhibiting and controlling effects of one growing part over other parts; and that after adult age is reached a state of balance is largely confined to the repair of natural loss. There still remains, however, considerable regenerative power; but this regeneration is never as perfect or well controlled in the adult as in the child. Stockard found also that regenerative growths following injuries to the adult may become morbid in degree, and lacking regulation give rise to tumor-like conditions.

Symptomatology.—There is found constipation in one who has always been regular; an increasing difficulty in getting the bowels to move; the passage of blood, or blood and mucus; and an imperative desire to move the bowels, with explosive movements of gas and mucus. At first, this necessity of moving the bowels more frequently than has been usual will occur occasionally through the day; but, later on, the desire becomes more frequent and urgent, and the patient has to rush to the toilet the first thing in the morning. As the disease advances he must get up at intervals during the night. There is always an indefinable feeling in the pelvis, accompanied by indigestion. Sometimes the stomach symptoms are so pronounced that one may be led to believe that the lesion is located in the stomach rather than at the other end of the canal. Hemorrhoids are nearly always associated with cancer of the rectum. Many patients seek relief for this simple condition, ignorant of the more serious lesion existing higher up. This explains why so many patients come to us with cancer who have been previously operated on for hemorrhoids, when unquestionably carcinoma existed at the time of the hemorrhoidal operation, but was not then discovered.

Cachexia, loss of weight, pain, are all terminal symptoms, and are seen, as a rule, only in very late stages of cancer. When the tumor is situated in the anterior wall of the rectum, bladder symptoms may be evident even though there is no involvement of the prostate or bladder. These symptoms can be explained as being due to pressure or a pulling of the tumor, or to a perirectitis due to the ulceration.

Diagnosis.—The diagnosis is made by the examining finger, or the proctoscope. As adenocarcinoma is the most frequent type of malignant tumor, the finger usually comes in contact with a round, nodular mass, with a crater-like ulcer in the center. Its appearance as seen through the proctoscope is that of a cauliflower growth with a central ulcerating surface. In addition one sees a bloody, mucoid discharge covering the mucosa below the tumor, which makes it difficult, at times, to see the growth. These tumors are not infrequently located on the upper fold of Houston's valve. At times nothing except a little blood will be seen to indicate the presence of a tumor, so that it might be easily overlooked during a proctoscopic examination. Infection of the bowel, causing nodular outgrowths, may be mistaken for cancer; but, as cancer is usually local and the infection commonly extensive, one should incline to the diagnosis of an ordinary infection rather than malignancy in this instance. However, a chronically infected bowel (as in two instances in the author's experience) may occasionally degenerate into carcinoma; and this makes the diagnosis between cancer and stricture very difficult at times. It is sometimes quite a puzzle to distinguish between a perirectal stricture and a sarcoma, because the mucosa is not always involved in either condition. One must always bear in mind that a secondary deposit from a primary tumor in the uterus may infiltrate the wall of the rectum and resemble a myoma, rather than an adenocarcinoma. Locating the primary tumor will clear up the diagnosis. Multiple diverticulitis of the lower sigmoid may closely simulate a carcinoma; especially as loss of weight, hemorrhage and cachexia are frequent accompaniments of diverticulitis, but the history of temperature, and long duration of the disease, together with proctoscopic examination, should be helpful in differentiating one from the other. Hyperplastic tuberculosis may be mistaken for carcinoma; but as primary tuberculosis of the bowel is rare, a lesion in the lung should help to determine this.

Treatment.—Too much emphasis on the necessity of an **early colostomy** in inoperable tumors of the rectum cannot be made. It allays pain, prolongs life, and enables the patient to attend to his usual occupations for some time. The operative results of the author are encouraging; about 26 per cent. are alive after five years. Unfortunately very few patients are seen in the early stages of the disease, so that taking it all in all the results may be considered good. The immediate mortality from operation is about 16 per cent. in patients who have not been selected.

X-ray or radium alone, so far as the experience of the author goes, is **not efficacious.** He has been using these methods, combined with surgery, for over a year, at St. Bartholomew's Hospital, New York, but with indifferent results.

CHAPTER XXVII

VISCEROPTOSIS OR SPLANCHNOPTOSIS; UMBILICAL AND EPIGASTRIC HERNIA

By Lewellys F. Barker, M.D., LL.D.
and
Maurice C. Pincoffs, B.S., M.D.

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VISCEROPTOSIS

Definition.—Visceroptosis is a term applied to the condition in which certain of the thoracic and abdominal viscera are situated at a level lower than normal. This situation of the viscera may be merely a part of a more general constitutional inferiority of congenital origin (*habitus asthenicus*), or it may be an acquired abnormality in a body type that was originally normal. In either case, visceroptosis, even of a marked grade, is often compatible with normal function of the viscera. In a considerable percentage of cases, however, abnormalities of function are present, which give rise to clinical symptoms. In a small number of cases, and those only of the extreme type, the ptosis of certain viscera may *per se* occasion painful sensations.

The patient with the asthenic habitus of congenital origin is characterized not only by visceroptosis but also by an unstable nervous system. Either the gastro-intestinal or the psychoneurotic symptoms may predominate in the individual case and disturbance in either sphere may react upon the other.

Etiology.—The congenital tendency towards a certain type of body structure, which includes visceroptosis as one of its stigmata, is perhaps the most important etiological factor to be considered. This body type has rather arbitrarily been considered by some as evidence of a degeneracy due to the influence of civilization. By others it is considered as evidence of the persistence of certain prehistoric racial characteristics of which carnivorousness is the most prominent. Certainly, there must be a foundation in the genotype to account for the peculiar phenotype that develops. It will be convenient to speak of this body-type as the *habitus asthenicus.*

In this type of body the mechanisms that normally retain the abdominal viscera at their usual levels are at a disadvantage; faulty posture, lack of muscle bulk, lack of muscle tone and lack of sufficiently developed fat pads in the abdomen conspire with congenital hypermobility of the organs (due to lack of completion of peritoneal fusions, etc.) to result in a descensus of the viscera. By some (Albu) the abdominal visceroptosis in these cases is supposed to be present from birth, but more recently R. H. Smith has stated that only the tendency is congenital, the visceroptosis appearing at about the age of puberty. Faulty posture is undoubtedly a factor which commonly aggravates the mechanical disabilities of this type of body make-up (Goldthwait). Childbearing, by the resultant impairment of the anterior abdominal wall, and, perhaps equally, by the strain it imposes on the nervous system, is of real etiological significance in the acquired type. Loss of weight, especially rapid loss of weight, has a great influence in aggravating the condition, just as the degree of chronic undernutrition is very apt to run parallel to the degree of visceroptosis.

As an important secondary factor in some cases must be included the wearing of tight corsets by women and of tight belts by men. Overweighting of certain portions of the gastro-intestinal tract with the digestive products (due to atony, dilatation, and stasis) occurs in visceroptosis and leads to still further descensus of the parts affected. This is especially true of the ptosed stomach and cecum. The importance of the secondary factors is best demonstrated in the cases of acquired visceroptosis seen frequently in women who have borne many children or who have become rapidly emaciated.

The etiological factors are best appreciated when one considers the normal mechanical supports of the abdominal viscera. These include: (1) The suspension of the viscera from the diaphragm and the factors supporting the diaphragm (vacuum in thorax, pericardium, cervical fasciæ to lower skull); (2) the fusion of the viscera with the posterior peritoneum; (3) the lumbar lordosis and the psoas muscle forming a shelf for the kidneys and narrowing the lower abdomen; (4) the tonus of the anterior abdominal wall, allowing it to accommodate itself to the varying bulk of the abdominal viscera at different phases of digestion, with preservation of a constant even tension that offers resistance to forward or downward displacement; and (5) the fat in the retroperitoneal tissues, the mesentery, and the omentum, supporting everywhere the heavier viscera and preventing undue dragging upon their peritoneal attachments.

The resultant of all these factors is "abdominal equilibrium" between the elements supporting the viscera and the force of gravity tending to displace them downward. Faults in any one or in several of the factors mentioned favor splanchnoptosis.

Symptomatology.—As has been pointed out, the cases of splanchnoptosis may be divided into two great groups from the standpoint of symptomatology: (1) Those presenting no symptoms (asymptomatic type); and (2) those presenting general or local symptoms, or both (symptomatic type).

The general symptoms presented by patients with the *habitus asthenicus* and visceroptosis are well known. They are those of asthenia, the patients tiring easily on physical or on mental exertion. They constantly complain of lack of endurance. There is often slight tachycardia and vasomotor disturbances are not uncommon. Many of those presenting the so-called effort syndrome, or neurocirculatory asthenia, are of the asthenic habitus. A large proportion of the patients also exhibit a labile psyche, no small contingent of the psychoneurotic group consisting of splanchnoptotics who complain of nervous dyspepsia.

In *gastroptosis,* the patients may complain of anorexia, of a feeling of fullness in the stomach or of subjective symptoms referable to changed secretions. Nausea, gaseous eructations and intestinal disturbances (constipation, mucous colitis) are frequent complaints. In addition the patient with gastroptosis who complains of symptoms usually emphasizes feelings of weakness, insomnia, headaches and depression. When the duodenum as well as the stomach is ptosed there may be marked disturbances of nutrition.

The subjective complaints associated with the *enteroptosis* consist of constipation, various kinds of painful sensations in the abdomen, and abdominal distention with protuberant lower abdomen. Many of these patients have their minds centered upon the abdomen and suffer from hypochondriacal depression. This depression is always worse during attacks of mucous colitis, so common in this condition. The constipation in enteroptosis may be either of the atonic or of the spastic type, though the latter is the more common.

In *hepatoptosis* with mobile liver some patients may complain of no symptoms at all, whereas others suffer distress from a feeling of weight and tension in the abdomen and have associated disturbances of digestion. Signs of venous obstruction may appear owing to kinking of the portal vein or of the vena cava. Occasionally the hepatoptotic patient may suffer from pains in the back that radiate toward the shoulder or toward the sacrum. Sometimes, too, the tug on the diaphragm, due to the low position of the liver, may cause respiratory or circulatory disturbances. If the gall-duct be kinked or the hepatic blood-vessels be partly occluded by a kink, secondary changes in the liver may follow with corresponding symptoms.

Ptosis of the spleen usually causes no subjective complaints, though the patient may feel the mobile organ in an abnormal position and be alarmed by this. Now and then a spleen is so mobile as to undergo partial torsion or complete torsion causing interference with the circulation and necrosis.

In *nephroptosis* there may or may not be subjective complaints on the part of the patient. Most often the displaced organ is discovered accidentally on abdominal examination by the physician. Some patients complain of a drawing feeling or of pressure in the region of the kidney and fear that they suffer from some serious disease. Even when a wandering kidney is discovered it is questionable whether the physician should inform the patient of the fact, for many of those patients easily become hypochondriacal and ascribe a serious importance to an anomaly that is usually of but little significance. If a patient has been told of a wandering kidney and is worried about it, he should be given strong reassurance by the physician that the anomaly as a rule is harmless; if local treatment is avoidable, all the better. In cases of extreme mobility of the kidney, however, sudden attacks known as "Dietl's crises" may occur, the patient suffering from violent colicky pains in the kidney region due to kinking of the ureter and to urinary obstruction. Such attacks are most often precipitated by bodily overexertion, by the lifting of heavy weights, or by bending or stooping. The attacks may be accompanied by vomiting, chills, fever and collapse; there may also be oliguria and sometimes hematuria. After the attack is over the patient notices a sudden polyuria with disappearance of the local swelling. Structures adjacent to the kidney may occasionally be compressed by a floating kidney. Among the organs that may be thus pressed upon are the duodenum, the bile-duct or the neighboring intestines.

In *cardioptosis,* or *drop heart,* there are often no symptoms referable to the heart itself, though some patients complain of a little breathlessness on exertion and notice that they have a rapid pulse, especially on excitement or on exertion. The low blood-pressure and the vasomotor disturbances associated with this condition have already been referred to.

In *general splanchnoptosis* with asthenic habitus it is common to find many signs of neurasthenia or of psychasthenia. The patients complain of headache, of urticaria, of periodic circulatory disturbances, periodic dyspepsias, nervous asthma, coryza vasomotoria, false angina, etc. The symptoms of nervous dyspepsia are usually in the foreground and are protean in form. They may include anorexia, glossodynia, cardiospasm, food idiosyncrasies, nausea, gastralgia, entralgia, abdominal hyperesthesia, chronic constipation, rectal disabilities, mucous colitis, intestinal fermentation, and the like. Many of these patients are vagolabile; some of them are vagotonic; and some are sympatheticotonic. They seem to be somewhat more prone than the majority of people to focal infections, as their powers of resistance to infection seem to be diminished. This perhaps accounts for the incidence of pulmonary tuberculosis, of duodenal ulcer, of appendicitis and of chronic arthritis in patients exhibiting the *habitus asthenicus.* Among mental defectives and among patients suffering from true psychoses the enteroptotic habitus is especially common as a part of a general congenital inferiority.

Diagnosis.—The *habitus asthenicus* is recognizable at a glance by the eye of the experienced clinician. The tall thin patients with long trunks, flat chests, acute epigastric angle, floating tenth ribs, and sunken abdomen with protuberance of its lower part tell the physician with what he has to deal as soon as he sees his patient undressed. On examination his first impression is confirmed by discovery of a hypoplastic drop heart, a narrow aorta, the objective signs of visceroptosis, small retroflexed uterus, etc., and these objective signs are usually further corroborated by the psychoneurotic phenomena manifest on conversation with the patient.

The objective signs of visceroptosis are easily discoverable by physical examination and by roentgenological studies. In *gastroptosis* the greater curvature may, on distention of the stomach, be found below the level of the umbilicus, the pylorus itself often being situated below the navel. The lesser curvature of the stomach is correspondingly depressed; when on distention of the stomach the pylorus is found between the xiphoid and the umbilicus or below the umbilicus, one can be sure that a gastroptosis exists. The condition is most easily recognized, however, on roentgenoscopic examination in the standing position. The body of the stomach is drawn out into a long tube that runs almost parallel to the spine, and the pylorus and the lesser curvature are visible at a low level, for *pyloroptosis* is usually present also. Such ptotic stomachs are usually atonic, but one does meet with gastroptosis without atony and one may see atony without any ptosis. The motility of the gastroptotic stomach is usually slightly delayed. On chemical examination there is frequently superacidity but not always. A dilatation of the duodenum in association with gastroptosis is recognizable in the x-ray by the finding of the "sausage-shaped duodenum" of Holzknecht. Reflex cardiospasm is by no means uncommon in cases of gastroptosis.

In *enteroptosis* the displaced intestines, especially a displaced cecum or a displaced transverse colon, can often be felt on palpation. The exact position of all parts of the intestines can however be determined best by means of x-ray examinations made after a contrast meal. For the determination of the position of the different parts of the large intestine, x-ray plates made after a contrast enema are very helpful. In ptosis of the transverse colon (*transversoptosis*) this portion of the intestine may be seen as a V-shaped or a W-shaped loop in an abnormally low position.

In *hepatoptosis* the dislocated liver is palpable as a large movable tumor in the right upper abdomen. If one palpates while the patient is standing, the liver may even be found in the lower abdomen, though in the recumbent position it can be pushed back into its normal place, except in rare cases in which it has become adherent at an abnormal site. In extreme instances of wandering liver the whole organ can be grasped between the two hands in the middle or lower abdomen and it may be possible to feel distinctly the incisure in the liver margin. One who feels an outspoken wandering liver for the first time is not likely soon to get over his surprise. When the liver is freely movable and easily replaceable there should be no difficulty in recognizing the nature of the mass. When, however, the liver is fixed by adhesions in an abnormal position this may be far less easy. Such a mass has been mistaken for pelvic tumor, for hydronephrosis, for mobile kidney, and for omental tumor.

A *wandering spleen* should not be mistaken for an enlarged spleen that is palpable below the costal margin without increased mobility. In simple wandering spleen the blood is unchanged.

In *nephroptosis* several degrees of mobility occur. When only the lower pole of the kidney is palpable on deep inspiration the organ is spoken of as a "palpable kidney"; if on bimanual palpation the kidney can be captured at the end of deep inspiration and held down, though not below the level of the umbilicus, the organ is spoken of as a "movable kidney"; if there be still greater mobility, so that the kidney can be pushed downward as far as the groin or medialward as far as the middle line, it is spoken of as a "floating kidney." Care must be taken not to confuse a hypermobile kidney with other masses that may resemble it, such as a large gall-bladder, an ovarian cyst, or an omental or intestinal neoplasm. It is probable that Dietl's crises, when they occur, often go unrecognized, for the attacks may be supposed to be due to renal colic, to bilary colic, or to appendicitis. When there is doubt as to whether right-sided pain is due to a Dietl's crisis associated with a movable kidney or to some other cause, an examination by a urogenital specialist with catheterization of the ureters will usually decide, for the pain of Dietl's crisis can often be reproduced between attacks by injecting fluid into the ureter of the same side. The patient recognizes immediately the identity of the artificially produced pain with the spontaneous attack. X-ray plates of the renal pelvis on the two sides after thorium injection will often show a slight dilatation on the affected side due to slight hydronephrosis.

The *drop heart* or *cardioptosis* is easily recognizable on physical examination and especially on roentgenoscopic or teleroentgenographic examination. The median position of the cardiovascular stripe, its narrowness, the small area of the heart in contact with the diaphragm, the lateral mobility of the stripe on change of position of the patient, the high level of the base of the heart, and the exaggeration of the second curve on the left side, are striking features on roentgenoscopic examination. In the teleroentgenogram the peculiar position of the heart, its small size, as well as the small size of the aorta, can easily be made out. Sometimes a soft systolic murmur is audible over the heart. The blood-pressure is usually low.

In the constitutional form of visceroptosis the determination of the so-called *splanchnoptotic index* of Becher-Lennhoff may be interesting. It is obtained by dividing the distance of the jugulum from the upper margin of the symphysis pubis by the minimal circumference of the abdomen and multiplying the result by one hundred, thus:

$$\text{Index} = \frac{\text{Jugulo-pubic distance}}{\text{minimal abdominal circumference}} \times 100.$$

In normal women the index in the recumbent position averages 75, in the standing position a little less, whereas in man it is less than 75. A higher index, for instance over 80, is met with in the *habitus astheni-* and in people with palpable kidneys.

In the acquired form of splanchnoptosis, seen in thin women who have borne many children and due to relaxation of the abdominal walls and the mesentery, a marked diastasis of the recti muscles can usually be demonstrated. One asks the patient when lying on her back to raise the head, when immediately a marked bulging appears between the recti muscles of the abdomen. On standing, such a patient will exhibit a strikingly pendulous abdomen owing to the atrophy and flaccidity of its walls. Sometimes the whole of the examiner's hand can be inserted between the recti muscles and the several organs within the abdomen can be grasped.

A description of the differentiation of symptoms due to ptotic viscera from those occurring in the various subacute and acute inflammatory conditions within the abdomen would lead us too far afield. It goes without saying that the physician should in every case try to make his diagnosis complete. The mere finding of splanchnoptosis does not mean that other conditions may not also be present; indeed, it is not at all uncommon to meet with inflammatory and neoplastic conditions in the abdomen of patients exhibiting splanchnoptosis.

Treatment.—In the treatment of visceroptosis we make use of measures that will restore normal nervous tone, of measures that will overcome the mechanical deficiencies, of measures that will combat the functional defects and, afterwards, of measures that tend to keep the patient in good physical and mental condition and that will prevent a recurrence of symptoms.

As has been said, the majority of visceroptotic patients are markedly undernourished and many of them are confirmed psychoneurotics. Often a **thorough rest cure** with general upbuilding, the **putting on of weight, massage, hydrotherapy, psychotherapy**, and, later, **occupation therapy**, will do more than anything else to restore the patient, who has become incapacitated, to a nearly normal if not to a completely normal life. In mild cases so radical a procedure is, of course, unnecessary, but in the severer forms of the disease, where the patient is thirty or forty pounds below normal weight and where the nervous and mental symptoms are marked, the shortest way out of the difficulty is to persuade the patient to enter a **hospital**, a **sanitarium** or a **nursing home**, where a scientifically conducted rest- and upbuilding-cure can be given. An intelligent **special nurse** is a *sine qua non*, particularly when food phobias exist, or when delusions of interpretation of symptoms have become established. Nothing in practical medicine is, however, more gratifying than the results obtainable in many of these cases when the physician is given a free hand, unmolested by the family or the friends of the patient, to control the rest, the diet, the exercise, the occupation, etc., for a few weeks, with utilization of all the modern physical and mental methods of therapy that are at his disposal. Simple methods are, however, preferable to complex methods. The physician who has secured the confidence of his patient by his thorough methods of examination and by friendly and sympathetic treatment can, as a rule, rapidly fatten these patients and do much to free them from their psychoneurotic torments without making use of anything but the simplest methods of rest, isolation, nursing, diet, and the milder forms of hydrotherapy, mechanotherapy, psychotherapy, and occupation therapy. In some sanitaria, too, many methods of treatment are made use of in the attempt to build up these patients. The authors have known of patients actually being worn out by multiple and superfluous therapeutic efforts. The symptoms of so-called nervous dyspepsia may indeed be very troublesome, but if one will begin with the **milk diet of Du Bois** and after a few days will go over to a sensible diet composed of all kinds of easily digestible foods, success will more likely attend his efforts than if he pays too much attention to the whims and caprices of his patients. Nor is it necessary to restrict a patient to a very dry diet as is often done in splanchnoptosis. These patients have some digestive disturbances at first no matter what diet they employ. It is therefore best as a rule to give an **all-round diet** and to teach the patients to bear with equanimity their symptoms until they disappear. Many of these patients swallow air, and nausea and vomiting may be troublesome for a time, but usually these symptoms can be gradually overcome. The secretory and motor disturbances of the gastro-intestinal tract may need a little pharmacotherapeutic attention, but the wise physician will **avoid any excess in the use of drugs.**

It is impossible to give general directions for the meeting of all the various symptoms that may be complained of. The **therapy must be individualized** in each case. Any organic changes found to be present must, of course, be given due attention. Very often, however, the existence of organic changes is a purely accidental association and treatment directed toward them has no influence upon the course of the "nervous dyspepsia," which yields only to treatment directed toward the general psychoneurotic state.

The greatest triumphs of rest- and feeding-cures are met with in the undernourished splanchnoptotics. Their condition has often been produced by overprotection of the stomach and fears regarding the ingestion of particular types of food. Not infrequently these patients will have more symptoms when on a very mild diet than when they are ingesting all kinds of foods, including those that are notoriously indigestible. The diet should contain plenty of vitamins as well as the staple articles of food. If a patient take some milk, some leafy green vegetables and some fresh fruit or fruit juice each day he will ingest all the vitamins that are necessary. Excesses of condiments are of course to be avoided, but some tea and coffee may be permitted, and a little wine or beer, especially with the principal meal, will often be found helpful. A **bitter tonic** before meals may increase the appetite when it is lacking. **Mild sedatives** may be necessary at night if insomnia be a troublesome symptom, though as a rule it is best to treat insomnia by physical and psychical rather than by chemical methods.

Of the measures used to repair the mechanical defects in splanchnoptotics, the gaining of weight and especially of fat comes first. Fat in the omentum and in the peritoneal tissues is the best of all supports for ptotic viscera. Next to fattening the patient comes strengthening of the anterior abdominal wall by means of suitable **calisthenic exercises.** If there are postural defects, they should also be corrected by the well-known measures now utilized by the orthopedists. If the patient should be compelled to be up and about before he can be made to gain weight and to correct his posture, some help may be given by means of **palliative corsets, binders, strapping,** etc. The authors rarely find it necessary, however, to use these mechanical appliances in the treatment of visceroptosis, and unless they are absolutely unavoidable they are certainly a nuisance to a patient. Now and then there may be a patient who requires some such appliance, but it is surprising how well one can do without them in by far the majority of the cases.

For some time, as is pointed out in the paragraph dealing with the history of the subject, surgical measures were made use of for fixing loose organs in position. These have now, and with right, been entirely abandoned, except, perhaps, for an occasional case in which it seems impossible to prevent Dietl's crises (due to kinking of the ureter) without surgical interference. Even here, however, fattening the patient and the wearing of a suitable support will in most cases make surgery entirely unnecessary.

In the treatment of the functional deficiencies associated with visceroptosis, measures for the overcoming of gastric hyperacidity and of chronic constipation are those indicated most frequently. When the stomach contents are hyperacid and the hyperacidity gives rise to subjective symptoms, it may be well to use a **blunting powder** after meals, at least for a time; any one of the well-known formulæ may be used. It is the authors' custom to give a quarter of a teaspoonful of the following powder a half hour after each meal to relieve pain due to gastric hyperacidity:

℞

Magnesiæ ustæ	10.0 grams	(154.32 grains)
Sodii bicarbonatis	10.0 grams	(154.32 grains)
Potassii carbonatis	6.0 grams	(92.60 grains)
Extracti belladonnæ	0.7 gram	(10.803 grains)

Such patients are often vagotonic and are benefited by taking **Trousseau pills** before breakfast each morning. The formula of Trousseau's pill is as follows:

℞
Extracti belladonnæ,
Folii belladonnæ..........................āā 0.3 gram (4.63 grains)
Extracti gentianæ q. s. u. f. pil. No. 30.

The authors begin by giving two pills before breakfast and increase the number until six or eight are taken each morning. After a while the dose may be gradually reduced and later the pills may be left off altogether.

When intestinal stasis exists, whether the patients complain of constipation or not, it is important to determine by **x-ray examination** the kind of constipation that exists, whether atonic or spastic, and also the region of the intestines in which accumulations occur. Suitable diets are then ordered and in addition one may make use of **mineral oil** as a lubricant and of **agar-agar, regulin, bran,** or **seeds of Psyllium** to give added bulk to the intestinal contents. An occasional laxative or purgative may be necessary. Now and then it may be desirable to give an **enema,** but the habitual use of purgatives and of enemata is most objectionable, and if the patient has formed a purgative or an enema habit the physician will do well to overcome it and to reëducate the patient to normal bowel movements obtainable by simple hygienic measures. Many constipated patients drink too little water; a **glass or two of water** on rising and the same amount an hour before the midday meal and an hour before the evening meal should be prescribed in such cases. In a few instances, actual organic obstruction may be found in the intestinal tract, in which event surgical measures may be necessary to overcome it.

If thin patients are fattened and if psychoneurotic patients are rested and reëducated to normal habits, the symptoms usually ascribed to splanchnoptosis will as a rule disappear. There are almost sure to be relapses, however, unless the patient is kept under supervision by the physician for a long time after the preliminary treatment. The **patient** should be instructed to **report at definite intervals,** the length of time between visits being gradually increased as the health becomes more firmly reëstablished. Definite instructions as to the mode of life should be given in writing to the patient and at his recurring visits the several points should be checked up by the physician to make sure that the patient is not straying from the right path. It may be necessary for a **partially protective régime** to be maintained for some time. For instance, after a rest cure it is usually desirable for the patient to remain on part rest for at least several weeks, taking breakfast in bed and remaining there until 10:30 o'clock in the morning, resting lying down for an hour or so after the midday meal, and retiring at 9 o'clock P. M., even though he be able to walk an hour in the forenoon and an hour in the afternoon. Later on, the amount of rest may be diminished and the activities gradually increased. The patients should be warned to avoid excesses of all sorts, especially the excessive use of tobacco, of coffee, of tea, and of alcohol. A life in the open air is highly desirable for these patients and they should be taught to sleep with their windows wide open provided they can be kept warm in bed. Regular exercises and relaxation and congenial occupation should be arranged for throughout the whole of life.

Pathological Anatomy and Physiology.—We are now familiar with a considerable body of facts bearing upon the pathological anatomy of splanchnoptosis and upon the pathological physiology of the ptotic organs.

The asthenic habitus includes: (1) Tallness and narrowness of the body; (2) small bones with gracile bony framework; (3) long trunk; (4) flat chest, drawn in at its lower part, with a very narrow costal angle, the lower ribs often being only a couple of fingerbreadths above the crests of the ilia; (5) tenth rib floating, or at least attached only by ligamentous instead of cartilaginous attachments (*costa decima fluctuans* of Stiller); (6) abdomen sunken inwards above the umbilicus and protuberant below that level; and (7) posture, usually that of fatigue, the head hanging forward, the shoulders carried forward and the back flat with obliteration of the lumbar curve.

In the thorax, the cardiovascular apparatus is elongated and narrowed, the heart often standing almost perpendicular as the so-called "drop heart" (*cardioptosis*).

The abdominal findings include, in varying grades, the following: The stomach is prolapsed, sometimes being of the hammock type and descending in a median position, but more often being of the so-called fishhook type with the concavity to the right, the whole of the organ lying to the left of the spine. The cecum lies either with its lower pole at the brim of the lesser pelvis or, in more marked cases, with the greater portion of its bulk within the lesser pelvis. The hepatic flexure of the colon is usually but little, if any, above the crest of the ilium. The transverse colon descends in a "V," or in a loop, to the level of the symphysis pubis, or even lower so as to extend into the lesser pelvis. The splenic flexure of the colon is usually in its normal position, showing very little descent. The liver, spleen, and left kidney are, as a rule, but little prolapsed, though in severe cases a "wandering" liver or spleen may be met with and the left kidney as well as the right may occupy a low position and be preternaturally mobile.

These findings are those commonly met with upon x-ray studies of the gastro-intestinal tract after a barium meal. The positions visible in x-ray plates may not be exactly those of the viscera on fasting, or those during the digestion of a full diet. However, they approximate such positions quite as closely as do autopsy findings, and differentiate the cases quite clearly from the normal.

In association with abdominal visceroptosis pathological changes of various sorts are frequently met with in the abdomen. These include dilatation of the atonic prolapsed stomach, duodenal dilatation, intestinal kinks, especially of the ilium and of the colon, mobile cecum, ulcerative and nonulcerative colitis, especially mucous colitis, adhesions in various regions, especially in the right upper quadrant and the right lower quadrant, the formation of new membranes, congestions of ptotic viscera due to kinking of their blood-vessels, hydronephrosis due to kinking of the ureter of a ptotic kidney, etc. Space will not permit further mention of these pathological conditions. For further descriptions of them textbooks of pathological anatomy should be consulted.

Many patients with splanchnoptosis are devoid of outspoken clinical symptoms referable to their disorder but in the severer forms symptoms of various sorts appear,—gastro-intestinal, urogenital or nervous.

The gastro-intestinal disturbances in visceroptosis depend upon disturbances of motility, upon disturbances of secretion, or upon both. These disturbances, in turn, depend in part upon the mechanical conditions present, and in part upon disturbed innervations of the vagal and sympathetic nervous systems. Similarly, the urogenital disturbances depend in part upon mechanical interference with the lumina of the blood-vessels and the ureters owing to kinking caused by hypermobile kidneys, in part upon pressure effects upon the urinary passages and genital organs, and in part upon disturbed innervations. The nervous symptoms have their origin partly in local conditions within the abdomen, and partly in the congenital inferiority of the nervous system as a whole that is so common in the *habitus asthenicus.* Forms of visceroptosis that have been asymptomatic for years may become symptomatic owing to disturbance in nervous regulation of the gastro-intestinal functions, less often owing to an actual increase in the degree of mechanical defect existing.

Historical Comment.—The recognition of visceroptosis by Glénard and of the asthenic habitus by Stiller marks an important incident in the long history of the attempts made by medical men in all the ages to discover a pathological basis for the symptom-complex that is popularly termed "nervous dyspepsia."

In this "dyspeptic syndrome" are included a host of symptoms—anorexia, food phobias, fullness after eating, nausea, abdominal pains, tenderness, flatulence, constipation or diarrhea, mucous colitis, etc. There have always been those who have been inclined to consider it as primarily psychic in origin and others who sought an explanation in pathologic anatomical states of various abdominal viscera, or in perversions of the physiological processes of digestion. It is interesting and a little discouraging to trace the history of this subject and to note one by one the various theories appearing and holding sway over the minds of men, and then being gradually abandoned in favor of some new hypothesis, but often reappearing in a new garb a few generations or centuries later. To mention only a few instances, it is rather amusing to see the "black bile" of Hippocrates now being reintroduced to the medical world by those who are enthusiastic over biliary drainage through the duodenal tube after magnesium sulphate has been introduced, to see the chronic peritonitis of Rokitansky and Virchow being heralded anew by Lane, and to note that the clysters of Molière's day, which were supposed to wash out from the bowels the infarcted humors (*succus melancholicus*), are even now used as a panacea at some of our well-known sanitaria. At the moment, colonic irrigation is zealously supplemented by regular intracolonic implantations of *Bacillus acidophilus* and other "friendly" bacteria. The state of the liver, of the bile, of the spleen, of the gut and of the female generative organs, congestion in the portal veins, peritoneal adhesions and abnormalities of position of the viscera have all been used at times as explanations of nervous dyspepsia and no doubt will be so used again.

Some physicians have always taken the middle ground and felt that in some cases presenting this symptom-complex of "nervous dyspepsia" the condition was primarily psychic in origin and that in others it was primarily due to local abdominal conditions. Boerhaave spoke of *hypochondria sine materie* and *hypochondria cum materie;* and in later times we have been told of "primary psychoneurotic states" and of "reactive psychoneurotic states."

The alterations of position of the abdominal viscera as a cause of "nervous dyspepsia" were, as far as we can find, but little mentioned before the nineteenth century, though de Haen, in 1768 and later, published wood cuts showing abnormal positions of the viscera and drew attention to the atypical symptoms thus engendered. In the early part of the nineteenth century more notice was taken when Esquirol described the prolapse of the transverse colon that he found in 33 of 168 mental cases, especially among melancholics. G. Fleischman noted the frequency at autopsy of anomalies of position of the intestines and notably of the large intestine and stated his belief that a number of these cases were referable to fetal developmental anomalies. Cruveilhier drew attention to the influence of corsets upon the form and position of the viscera. Rokitansky described the changes in position due to adhesions. Virchow in 1853 gave to this condition the name of *chronic partial peritonitis* and called it one of the most important diseases, one to which the attention of physicians could not be too much drawn. He pointed out, also, the greater frequency of adhesions affecting the colon and believed that the abnormal positions of the gut seriously disturbed its function and gave rise to symptoms. Such symptoms, Virchow stated, might well explain the appearance of abnormal nervous and mental phenomena.

These earlier observations were based upon postmortem findings, but Rayer brought the subject of visceroptosis into the field of clinical diagnosis when he described the palpation of the movable right kidney. This observation led to a flood of publications. The most varied and the most severe symptoms were attributed to the overmobility of organs. The nervous and dyspeptic phenomena were especially noted in patients with movable kidneys and, since no other pathological process was discoverable besides the palpable kidney, it was felt that this must be causing the symptoms by mechanical or reflex means. The interest in this subject reached its height when, in 1878, Martin of Berlin performed the first nephrectomy for movable kidney and in 1881 Hahn of Berlin published an account of the first nephropexy.

One might digress at this point to state that with these dates begins the rather sorry story of the surgical treatment of neurasthenia. With freedom to enter the abdomen with impunity came an era when all the theories, ancient and modern, as to the causation of the nervous dyspepsia syndrome by disorders of the abdominal viscera, were joyously adopted by the surgeons and put to the surgical test. It has now been proven rather conclusively that removing the ovaries, womb, kidneys, colon, appendix, gall-bladder and portions of the stomach will not cure this type of neurasthenia; nor will short-circuiting of the hollow viscera nor stitching them up into supposedly normal positions do so. Many surgeons have learned these lessons and are inclined now to tell these patients that they are suffering merely from "nerves" and send them home. Valuable knowledge has been gained therefore by this era of surgical experiment. And after all some wars have cost an equal amount of suffering with no gain at all.

It was not long before it was noted that the prolapsed right kidney was often associated with other structural abnormalities. In 1881 Landau drew attention to the frequency with which a movable kidney could be demonstrated in multiparæ with relaxation and bulging of the anterior abdominal wall below the umbilicus. More difficult to explain was the frequent association of *ren mobile* with the dilated and prolapsed stomach, which had been described by Kussmaul.

A great advance was registered in 1887 with the appearance of F. Glénard's publication on enteroptosis. The significance of his contribution is in his view of the prolapsed kidney as constituting not a disease in itself but merely one feature of a more general condition. Many details of Glénard's theories failed to meet with general acceptance but in the above-stated respect he inaugurated our present conception of the nature of visceroptosis. Glénard associated in one and the same disease-picture the relaxed abdominal wall, the mobility and descent of the right kidney, the prolapse of the stomach, and the ptosis of the colon. His explanation of the pathogenesis was an entirely mechanical one and was somewhat fantastic. The primary factor was, in his opinion, a descensus of the loosely attached hepatic flexure, due to trauma, repeated childbearing, typhoid fever, etc., which entailed a descent of the right kidney, the duodenum and the rest of the colon. Kinking of the gut led to gastric stasis, dyspepsia, etc. The whole condition he thought should be looked upon as an *entité morbide* with dyspeptic symptoms: the neurasthenic symptoms he believed to be purely secondary.

Though many were ready to believe that now at last, after Glénard's papers, the true pathology of nervous dyspepsia had been found, others, and among them Stiller, took a somewhat different view. Stiller pointed out the weakness of Glénard's mechanistic views and summed up the evidence in favor of a congenital origin of the visceroptosis that Glénard had described. He pointed out that these ptotic abdominal conditions are so regularly found in patients of a certain body form that from the external appearance of the persons alone the existence of visceroptosis may be accurately predicted. He described the visceroptotic habitus and coined the term *asthenia congenita universalis* to describe the clinical picture presented by these patients with labile irritable nervous systems, digestive neuroses and a constant physical type characterized by a gracile skeleton, a long thorax, thin relaxed muscles, thin panniculus, fluctuating tenth ribs and varying grades of visceroptosis.

Since Glénard's and Stiller's contributions interest in the subject of visceroptosis has led to much work along four main lines: (1) Amplification of Stiller's views as to the relation of body-type to visceroptosis; (2) study of the mechanical factors involved in the support of the viscera in their normal position; (3) investigation of the anatomical changes in visceroptosis; and (4) observations on the disturbances of function occurring in visceroptosis, and, in particular, on the atony and spasticity of various portions of the digestive canal with resultant stasis.

UMBILICAL HERNIA

Umbilical hernia may occur either in the child or in the adult.

Infantile Umbilical Hernia.—In the new-born child it may be due to *failure of the umbilical cord to obliterate* so that at birth a cystic swelling is present at the fetal end of the cord and may contain an intestinal loop. Cases of this sort have been described by Sheen, by D'Arcy Power, and by Reed. In such instances, a **radical operation** should be done at once. If this be impracticable, the cord should be ligated at a point distal to the hernial tract and the radical operation done later.

Another form of infantile umbilical hernia is that known as *amniotic hernia.* Occasionally there is a defect of skin around the umbilicus, the skin being replaced by amnion, which is reflected from the umbilical cord upon the abdomen. Into such an amniotic umbilicus a hernial protrusion may occur as a result of the intra-abdominal pressure and cases of this sort have been described by Stewart, by Sanderson and by Yates. It is possible that such children might be saved by **plastic operations** done immediately after birth.

Other forms of infantile umbilical hernia may be due to *congenital nipping off of an umbilical hernia protrusion* or to *tension of the umbilicus* with serous fluid in ascites or tuberculous peritonitis.

Adult Umbilical Hernia.—In adults umbilical hernia is much more common in the female than in the male, occurring especially after the abdominal distention due to pregnancy. It is also more common in fat people than in the thin. Such an adult umbilical hernia may contain a bit of omentum or a loop of the bowel, either of which may become incarcerated. The operative procedures necessary for the treatment of umbilical hernia are given in detail in Cullen (reference cited in Bibliography).

EPIGASTRIC HERNIA

Definition.—A hernia lying at the linea alba in the epigastrium is known as epigastric hernia. It may consist of fatty tissue only, or it may contain omentum or omentum and intestine.

Etiology.—Epigastric hernia appears to depend upon a congenital weakness of the abdominal wall and its fascia. Patients who suffer from epigastric hernia not infrequently exhibit also some other form of hernia (inguinal, femoral).

INCIDENCE.—It is a relatively rare form of hernia making up only 1 or 2 per cent. of all the recorded cases of hernia, though doubtless many of the cases are overlooked and the incidence may in reality be somewhat greater. It occurs far more frequently in men than in women and is commonest among laborers between thirty and forty years of age.

Symptomatology.—An epigastric hernia may develop insidiously and increase very slowly in size, or it may appear suddenly and exhibit at once the symptoms of incarceration. Many patients suffer from digestive disturbances for years before the real cause of their symptoms is discovered in an epigastric hernia. The patients complain of paroxysms of pain in the epigastrium, which are exacerbated by physical exertion or by the ingestion of food. They usually attribute their symptoms to stomach trouble. Pressure in the epigastrium increases the pain and the intensity of the pain often varies with a change of position. Vomiting may occur at the height of an attack.

Diagnosis.—Many cases of epigastric hernia are overlooked because the physician does not think of the possibility of its existence. The diagnosis is usually easy if one thinks of epigastric hernia as a possible cause of the symptoms and palpates carefully the linea alba. The gastropathies most frequently confused with epigastric hernia are ulcer of the stomach, cancer of the stomach and gastritis. Paroxysms of pain due to epigastric hernia have also been mistaken for gall-stones, and for renal colic.

Treatment.—The treatment of epigastric hernia is **surgical**, and it is important that the patient should not neglect the condition, for incarceration sometimes occurs and a fatal outcome has more than once been reported. Surgical treatment is very satisfactory in most cases, though many relapses have been reported. In many cases it is not necessary to open the peritoneal cavity at the operation. Capelle asserts that when true stomach symptoms are present in association with hernia in the linea alba, an **exploratory laparotomy** should always be done, for in his opinion an epigastric hernia has often been held responsible for symptoms that were in reality due to lesions within the abdomen.

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CHAPTER XXVIII

FLATULENCE

By Walter C. Alvarez, M.D.

Symptomatology, p. 753.
Etiology, p. 754:—Greater swallowing of air, p. 754; Slowing or stoppage of the intestinal current, p. 755; Interference with starch digestion, p. 755; Interference with intestinal absorption, p. 755; Interference with circulation, p. 755; Interference with exhalation in lungs, p. 756.
Diagnosis, p. 756.
Treatment, p. 756:—Drugs, p. 757.

Flatulence is one of the commonest symptoms complained of by the dyspeptic. As such, it deserves far more study than it has received in the past.

Symptomatology.—Clinical Physiology.—We must first concern ourselves with the question: Whence comes the gas?

(1) To begin with, much of it is swallowed. Most of the analyses made of the gastric gases show that they consist mainly of atmospheric nitrogen. The normal stomach always shows a gas bubble which remains in the fundus when the individual sits or stands. After many years spent in radioscoping stomachs, the author is satisfied that an actual distention of the stomach, such as most people think they have, is extremely rare. Their distress may be real and they may be sure that they are bloated, but the roentgen ray often shows that there is no increase in the abdominal gases.

Physicians must not make the mistake of thinking that the gas complained of by their patients is being produced by fermentation in the stomach. That is rarely the case for a number of reasons. Ordinarily the food leaves the stomach too soon for such fermentation. Even with the marked stasis which we see with pyloric ulceration there is often little gas production. Even a small amount of acid restrains the growth of the average bacterium, and the swallowed oxygen keeps the anaërobes from going to work. Several investigators have shown, moreover, that gastric contents left in the incubator for some time give off very little gas.

(2) Much gas is undoubtedly formed in the bowel by the fermentation and putrefaction of foods. Most of this must take place in the right half of the colon, the only place where we find the necessary combination of stasis with liquid material and plenty of bacteria. Boycott and Damant[1] found that goats produce from 10 to 30 c.c. (2.71 fluidrams to 1 fluidounce) of hydrogen and methane per kilo (2.71 lbs.) per hour. An

average goat of 20 kilos produces 9 liters (2.377 gallons) of such gases a day. Fries[2] states that a grown steer produces from 100 to 250 liters a day of methane alone. These animals, of course, have large reservoirs designed for the slow fermentation of coarse foods.

(3) Some carbon dioxid is supposed to be produced in the duodenum by the action of the gastric hydrochloric acid upon the alkaline carbonates of the pancreatic juice.

(4) Carbon dioxid and oxygen are excreted from the blood into the lumen of the stomach and bowel. This exhalation has been demonstrated clearly by a number of investigators who have shown that if measured amounts of air are put into a stomach or a loop of bowel, the air loses oxygen and gains carbon dioxid. It may be that some of the accumulations of gas which appear suddenly in nervous people arise in this way.

The next question is: How are these gases eliminated in health, and why is a normal person unconscious of their presence? The answer is that, for the most part, they pass rapidly through the bowel wall and into the blood, whence they are excreted by the lungs. Yllpo[3] showed that his stomach could absorb 12 c.c. of oxygen per minute. Carbon dioxid disappeared so rapidly that it was difficult to measure it. When he injected large amounts of air into his stomach, he had flatulence because the nitrogen was not absorbed and had to be passed as flatus. After injecting oxygen he had a little flatulence and after carbon dioxid none.

Kader[4] showed clearly that when the mesenteric circulation is impaired, the normal exhalation of gases from the bowel is slowed or stopped, while the passage from the blood into the bowel may be so great as to burst a loop which has been tied off.

Etiology.—The facts brought out in the foregoing discussion should make us look for flatulence in those conditions which lead to:

(1) A greater swallowing of air.
(2) A slowing or stoppage of the gastro-intestinal current.
(3) An interference with the prompt digestion of starches.
(4) An interference with intestinal absorption.
(5) An interference with the intestinal circulation.
(6) An interference with exhalation in the lung.

Actually we do find flatulence in just these conditions.

A GREATER SWALLOWING OF AIR.—This is brought about generally by the efforts of the patient to relieve himself of gas. Whenever a person belches noisily for minutes at a time, we may be sure that he is swallowing the gas as fast as it is coming up. Ordinarily this air is gulped down to about the level of the heart, but occasionally some will be forced into the stomach, and when that is brought up, the patient is happy for a few minutes. Although these air-swallowers are generally neurotic individuals, and although they can sometimes be induced to stop their belching over night, it must be remembered that most of them developed the habit in an effort to rid themselves of an uncomfortable feeling about the cardia. This feeling of tension seems often to be due to a disordered gastric peristalsis and this in turn is most commonly due, particularly in elderly women, to a diseased gall-bladder. Another common cause is a disordered heart action.

In many cases the roentgen ray examination shows that the gas which the patient is endeavoring to bring up is not in his stomach but in the splenic flexure where he could not get it if he belched for a long period. The author has often found hemorrhoids associated with this distention of the splenic flexure and there may easily be an etiological relation between them. The author tells these belchers that they are "scratching" themselves with the air, and that the best thing for them to do is to restrain themselves voluntarily, or by carrying a cork between the teeth until the desire passes off.

A SLOWING OR STOPPAGE OF THE INTESTINAL CURRENT.—It is well known that intestinal obstruction can lead to the severest forms of meteorism and even the voluntary restraint of a little gas in the rectum may lead to the rapid accumulation of distressing amounts. Furthermore, we know that constipation is a common cause of flatulence. The gas tends to accumulate above the fecal plug; it sets up violent peristalsis, and causes distress until the bowels move.

INTERFERENCE WITH STARCH DIGESTION.—A few people seem to be limited in their ability to digest starches, but in most of these cases, the real difficulty is that the starch grains have not been made sufficiently accessible to the amylases of the digestive tract. The cooking may have been insufficient to rupture the cellulose capsules; a heavy layer of grease may have been added; mastication may have been largely omitted, or too much rough cellulose may have been added to the diet. The food experts for the warring nations learned a few years ago that it did not pay, from the standpoint of nutrition, to leave in with the flour more than a certain percentage of bran because it interfered so much with the digestion of the starchy part of the grain.

Man has no ferment for the digestion of cellulose, and it is probably for this reason that green fruits, salads, and many of the green vegetables are so flatulent. Beans are flatulent, partly on account of their tough capsules, and partly on account of certain toxic oils. Similar drug-like substances are probably responsible for the flatulent tendencies of many melons, tomatoes, cabbages, peanuts, onions, etc.

INTERFERENCE WITH INTESTINAL ABSORPTION.—Purgatives may cause flatulence, perhaps by interfering with the absorptive powers of the mucous membranes, by altering the motility of the bowel, and by altering the circulation so that the gases cannot be carried to the lungs.

INTERFERENCE WITH THE CIRCULATION.—One of the commonest causes for flatulence is a failing heart. The resultant passive congestion not only interferes with the normal removal of gas from the bowel but it can even lead to increased exhalation of carbon dioxid into the tract.

High blood-pressure with or without sclerotic changes in the mesenteric vessels is also a common cause. Another disease which might interfere with the intestinal circulation is cirrhosis of the liver, and some authors enumerate flatulence amongst the symptoms of that disorder.

Common colds, especially in their prodromal stages, tend to upset the bowel and produce gas. Just why this should be we do not know, but it seems probable that the functions of the mucous membrane are disturbed by toxins or by bacteria.

Interference with Exhalation in the Lungs.—In pneumonia, meteorism may be so severe as to bring on death. The accumulation of gas may be due partly to the impairment of respiration and partly to a toxemic paralysis of the bowel. As one would expect, also, sudden accumulations of gas appear sometimes with attacks of asthma.

Diagnosis.—The sufferer from flatulence deserves a complete and careful examination. The history may point to a diseased heart, gall-bladder, or appendix; or the trouble may be traced to a dysentery, an asthma, a dietetic idiosyncrasy, a constipation, a too frequent use of drastic purgatives, a great fondness for chewing gum, or a nervous breakdown.

The physical examination may show an absence of teeth, a diseased lung or heart, a tender gall-bladder, a mass of hemorrhoids, a pelvic tumor, or what not.

Roentgen ray examination will help in ascertaining the presence of gas in the abdomen, the presence of ulcers, gall-stones, intestinal obstruction, diverticulæ, constipation, etc.

The examination of the stool will show the extent of mastication, the character of intestinal digestion, the presence of excessive amounts of cellulose or starch, or the presence of abnormal bacteria or parasites.

Treatment.—This depends naturally upon the cause found. The patient may get more help from **digitalis with rest**, from **nervous sedatives**, from a **cholecystectomy**, a **hemorrhoidectomy**, or the **removal of a fibromatous uterus** than from all the diets and carminatives in creation.

Most physicians prescribe as nearly as possible a **starch-free diet**, but a careful study of these cases, extending over many years, has convinced the author that it is much better to aim at the **removal of the cellulose** which so often is the cause of the poor starch digestion. If the raw fruits, salads, and greens are left out, if the bread is white, and the beans, peas, and tomatoes are put through a strainer, the patient's stool will generally change from a coarse, fermenting mess, full of starch, to a smooth paste in which very few starch grains can be found. With this change in digestion there is often associated a prompt and gratifying cessation of the symptoms. If this cellulose-poor diet does not help the patient, one can be sure either that the cause for his trouble is to be found outside the digestive tube, or that it is beyond the reach of purely medical help.

If any particular kind of starch is found to be hurtful, it should be left alone. Sugar in concentrated form should be avoided. It is one of the end products of starch digestion and when added in excess must tend to stop the process.

Coffee seems to produce flatulence in some people and that should be inquired into. Gum chewing should be stopped. Constipation should be regulated, if possible, by enemas, by the mildest of laxatives, or by paraffin oil. The latter often produces flatulence, and for that reason may have to be given up. A little calcined magnesia in the morning works nicely for many people.

DRUGS.—People who belch are generally relieved most by taking a little **soda** or **magnesia**. This reacts with the acid of the stomach to form carbon dioxid which is then brought up with great pleasure. It is hard to say just why this should give so much relief when one can see with the roentgen ray that the man's "Gas blasé" was normal to begin with, and if anything, a little larger when he finished. Some change is probably brought about in the gastric peristalsis which makes it less distressing to the patient. **Charcoal** is often given with the hope of absorbing the gas, but it can do that only when it is dry, and besides, few of the belchers have any excess gas in the stomach to be taken up.

The various carminatives may be used to advantage when the bowel is actually distended with gas. One of the best is **peppermint,** the oil or essence of which may be given in a little hot water or whisky, or on a lump of sugar. **Oil of anise and ginger** are old family remedies. **Asafetida** may be given by mouth or in an enema. **Spiritus etheris nitrosi** works nicely for some people. The **pulvis aromaticus** of the National Pharmacopeia can be prescribed alone or in combination with a little sodium bicarbonate or magnesia. Five or 10 grains (.324 to .65 gram) can be taken as needed in hot water. **Enemas** will give the most prompt and complete relief if the gas is low down or held back by a fecal plug.

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CHAPTER XXIX

EMBOLISM AND THROMBOSIS OF THE MESENTERIC VESSELS

By Walter C. Alvarez, M.D.

Incidence, p. 759; Etiology, p. 759; Symptomatology, p. 759:—Acute, p. 759; Chronic, p. 759—Diagnosis, p. 759; Treatment, p. 759.

Incidence.—The disease is seen most commonly in old men, although many cases have been observed in children. Trotter[1] collected 360 cases for his review of the subject in 1913.

Etiology.—First must be mentioned age. Valvular lesions of the heart supply emboli; hepatic cirrhosis with portal stasis favors thrombosis. In the young, acute appendicitis and pelvic infections may be the origin of the trouble.

Symptomatology.—The cases may be divided into acute and chronic.

Acute.—The patient is seized with agonizing abdominal pain. There follow nausea, faintness, vomiting of bloody material, often diarrhea with tarry stools or bright red passages, collapse, distention, tenderness and rigidity of the abdomen, with signs of intestinal obstruction, coma, and death.

Chronic.—Twenty-eight of the patients in Trotter's series gave a history of gastro-intestinal disturbances antedating their acute attack. They had flatulent distention, heart-burn, attacks of nausea and vomiting, diarrhea, constipation, and colicky pains. It is hard to say whether or not such symptoms should be ascribed to the arteriosclerosis, however, because these people so often have heart, kidney or liver disease which can also produce digestive upsets.

Diagnosis.—This is extremely difficult, as can be seen from the fact that only 13 out of 360 cases were recognized before operation or autopsy. When called to see a patient with agonizing pain coming suddenly in the upper abdomen, one must think of embolism or thrombosis if there are signs of embolism or serious arterial disease elsewhere.

Treatment.—The only hope for these patients lies in **immediate resection of the gangrenous bowel.** The mortality is naturally high, but out of 69 operated upon, 25 recovered.

REFERENCE

1. Trotter. Embolism and Thrombosis. Cambridge University Press, London, 1913.

CHAPTER XXX

NEW GROWTHS OF THE INTESTINES

By Thomas R. Brown, M.D.,
and
Ernest H. Gaither, M.D.

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Etiology.—At the present moment we possess no definite knowledge of the true etiologic factor or factors which have to do with malignant tumor formation. Various processes,—continued irritation or inflammation from mechanical, chemical, thermic or parasitic influences, or the displacement and irregular growth of embryonal tissue—have, at frequent intervals, been suggested as predisposing causes.

Heredity and family predisposition have always occupied a prominent place in the discussion of cancer or tumor etiology, but the case has not been proven. All are agreed as to the age incidence, namely, that most cases are met with in those past forty years. Sarcomata are more likely to occur in the young than carcinomata and statistics prove that intestinal tumors are found more frequently in the male sex, but no plausible reason has been advanced therefor.

Inasmuch as most tumor growths are located in the rectum, cecum and flexures, internal trauma is assigned a rôle in tumor production. It is most interesting to note that only about 5 per cent. of all intestinal cancers are found in the small intestine, while 80 per cent. occur in the rectum and 15 per cent. in the flexures, cecum and colon; also, that practically all growths of the intestine are primary in origin and show a markedly lessened tendency to metastasize as compared to tumors elsewhere; however, an occasional exception is noted in those rectal malignant tumors which may provoke secondary manifestations in the liver and gall-bladder, the large secondary growth overshadowing the small primary mass. Approximately from 8 to 11 per cent. of all carcinomata are found in the intestinal tract.

It has been claimed by some authorities that intestinal polypi and chronic ulcers show a tendency at times to undergo malignant degeneration. Malfunction of internal secretions and senility have received consideration as etiologic factors in tumor growth.

Symptomatology.—Clinical History.—The mode of onset in abdominal tumors is variable in different individuals; for instance, the first manifestation may be functional disturbance of the intestine due to interference with motility because of encroachment by the growth upon the intestinal lumen. This may be evinced by a feeling of abdominal fullness, distress and gaseous distention; also colic, paroxysmal and intermittent, may be the first intimation of a disturbed physiology. It is interesting as well as important to note that in the intestine, as in the stomach, the first onset of symptoms may be attributed to a dietetic indiscretion. With progression of the intestinal stenosis, secondary manifestations appear, such as loud borborygmic rumblings, often accompanied by visible and localized intestinal peristalsis; in the early stage the gaseous rumblings may be detected only by stethoscopic or direct auscultation of the abdomen. In a certain percentage of those cases where the onset is apparently produced by dietary indiscretion, a long latent period may follow the first acute attack, while subsequent intermittent attacks are followed by persistent and continuous symptoms.

The gaseous sensations, colicky pains, or both, may be generalized or localized or may radiate; as a rule, if the flexures are involved, the discomfort and pain are localized, depending necessarily upon the degree of stenosis; if the small intestine is involved the symptoms will most likely be situated in the region of the umbilicus; while if the large bowel exclusive of the fixed portions is the seat of disease, any region of the lower abdomen may be the site of complaint. Radiation of pain is not unusual; that is especially true of rectal carcinoma. It is important to note the relationship between bowel movement and the production or alleviation of discomfort or pain. Persistent tenesmus, as well as the radiation of pain to the back and loins, suggests localized rectal involvement.

Posture, relative to temporary intensification or alleviation of symptoms, may assist in locating a growth. As the stenotic process increases, with subsequent progressive narrowing of the tube, the colicky pains, borborygmi, visible persistalsis and constipation increase in severity and persistence. Should the process progress to complete occlusion, the well-known symptoms of intestinal obstruction, toxemia, fecal vomiting, collapse and death, will be the outcome.

Sudden, severe and persistent intestinal colic, without palpable tumor or disturbance of function, may be an early symptom of intestinal tumor formation. Again, an individual of carcinomatous age, who has been free of intestinal disturbance, but develops sudden persistent and unaccountable abdominal discomfort or pain, should be considered potentially malignant until proven otherwise.

Diarrhea as an introductory symptom of carcinoma of the intestine, or alternation of constipation and diarrhea is not unusual. The diarrhea may be due to enteritis and colitis produced by the irritative action of the growth, or putrefaction and fermentation because of the damming back of the fecal column. Where constipation is superseded by diarrhea, the latter may be the result of an ulcerative process tunneling through an obstructive tumor mass. It is rather an exception to meet with gastric disturbance in the early stage; however, as the obstructive process progresses and intoxication appears, there are loss of appetite, eructation, nausea, vomiting,—the latter first consisting of gastric contents and later of fecal material. The gastric contents may be hyperacid, normal, or, in the late stages when toxemia supervenes and the systemic resistance is diminished, subacid or anacid.

Obstruction in the ileocecal region, at times reflexly, interferes with the motor power of the stomach, restricting its emptying power; on the other hand, if the tumor is further along the intestinal tract, no such result is usually produced.

The discovery of an objective pressure pain point where subjective sensitiveness only has been previously noted is of great value. In a certain percentage of cases Nothnagel has ascribed this finding to a mild circumscribed peritonitis, and his assumption is doubtless correct.

A history of local or generalized abdominal distention is often elicited, is always worthy of note and warrants close study of the abdomen under various conditions and at different times.

The relationship between food intake and pain frequently is most misleading. The authors have known of cases where the ingestion of food, regardless of its character, produced intestinal colic; in such cases, if the colon is high or if the obstruction is in the region of one of the flexures, one is erroneously led to suspect a gastric lesion. The irritability of the intestine because of obstruction producing secondary results such as stagnation and colitis, causes it to be an easy prey to reflex influences; thus, when food enters the stomach and stimulates peristalsis, this same procedure is instituted almost immediately in the intestine by reflex influences originated in the stomach. Again, there are many cases where pain does not occur until four or five hours after meals.

The colics produced by tumor obstruction, because of their severity, tendency to nocturnalism, variability and irregularity as regards time and anatomical location may be mistaken for kidney, ureteral, liver, gall-bladder or appendix attacks. Intestinal colic is not likely to be preceded or accompanied by chills and fever and the characteristic symptoms which attend colic of the other abdominal organs. In malignancy of the intestine, as in all other pathologic conditions, we meet cases with unaccountable and atypical manifestations; instances have been noted where no attacks of colic have been observed during the course of the disease, while other cases presenting colic as one of the earliest, most persistent and severe symptoms will at operation show a lesion not at all commensurate with the functional disturbances. In these latter cases this atypical syndrome is undoubtedly due either to the irritability and hypersensitiveness of the bowel at the point of involvement, resulting in marked and oft-repeated spasm, or to the anatomical location of the growth, whether fixed or anchored because of an extension to intra-abdominal parts external to the lumen or wall of the intestine. In these cases colic may appear quite a long time before evidence of stenosis is produced; therefore, when complaint of vague, irregular, persistent or intermittent attacks of abdominal colic is made the possibility of intestinal neoplasm should be considered.

The following type of case is most puzzling and often offers a problem impossible to solve. The patient complains of mild, irregular, atypical but persistent digestive symptoms, not sufficiently distinctive to be classified as of stomach, intestine or reflex origin; of gradual loss of appetite and weight; and of anemia. Suddenly in the absence of a dietetic error, an intestinal stoppage occurs, with the various manifestations of acute intestinal obstruction. Frequently the only evidence of malignancy in such cases is the presence of occult blood in the stool.

The diagnostic importance of gastric symptoms as a result of tumor formation in the duodenum must not be overlooked; gastric dyspepsia, pain, discomfort, nausea, vomiting, and pyloric occlusion are prone to determine the pylorus as the seat of the lesion. Cases of proctitis, ulcers, hemorrhoids and fissures should be watched very carefully for future developments. In the absence of these conditions, one is naturally suspicious of tumor formation when irregularity of the bowel, diarrhea, constipation, or alternation of both, small caliber of stool, tenesmus, mucus, pus, blood, and pain or discomfort in the lower abdomen are encountered. Cachexia, anemia, loss of weight, general asthenia and incontinence are later manifestations of rectal involvement.

Symptoms of motor and sensory disturbances, such as fullness, distress, colic, constipation and diarrhea, may accompany benign tumors, but cachexia is a salient feature of malignancy only.

PHYSICAL FINDINGS.—*Observation.*—With rare exception, particularly if the process has been present for a prolonged period, a marked loss of weight is noted. A history of sudden loss of weight with normal or excessive appetite should arouse immediate suspicion of malignancy. Loss of weight, particularly if accompanied by anorexia, sitophobia or both, and anemia, are usual symptoms of the disease. While a certain percentage shows cachexia at fairly early stages, it is a late symptom in the majority of cases. Observation of the patient's general attitude will disclose asthenia, and in the late stages, edema. Toxemia may be revealed by the patient's mental state, and the writers are convinced that there is a close relationship between this condition and cachexia.

Observation (Local).—In the early stage of the disease no local manifestations, as a rule, are observed, but when obstruction, spastic or organic, occurs, general or localized changes of the abdominal contour are noted; there may be general enlargement indicating a diffuse intestinal gaseous distention, or it may be localized, suggesting a limitation of the gaseous dilatation to a circumscribed loop of intestine. Later visible peristalsis is noted and this, similar to distention, may be general, localized, mild or severe. It is not unusual in advanced cases to note anastalsis, or retrograde peristaltic movements. Peristaltic waves of either type, if not visible, may often be made so by striking the abdomen with a cold, wet cloth or applying deep pressure and massage; however, thick abdominal walls may preclude the possibility of witnessing this condition. Often the patient calls attention to the fact that visible peristalsis is accompanied by colicky pain. Tumor formation may be so large as to cause a mass to be visible on inspection.

Palpation.—In order that palpation may yield the fullest amount of information, it is advisable that the bowel be thoroughly emptied by laxatives, and, if necessary, to reinforce this procedure by bowel irrigation; the application of heat to the abdomen for some time or placing the patient in a hot bath is also of value. If these precautions are not taken, deceptive findings may lead to false conclusions, as incarcerated gas in an intestinal loop or hard fecal masses which easily simulate intestinal tumors. One of the difficulties encountered in this procedure is the extreme movability of the tumor, particularly if located in the small intestine, transverse colon and, to a lesser degree, the upper flexures. It is practically impossible to palpate a rectal carcinoma through the abdominal wall, and at times a mass in the sigmoid may be so far back toward the posterior wall that it cannot be felt; the descending colon usually does not present such difficulties. On account of being far back and posterior to the spleen, tumors of the splenic flexure cannot be palpated; here the patient should breathe deeply and the same procedures relative to change of the patient's posture, etc., should be adopted as when palpating for the kidney or spleen. The transverse colon is extremely movable and tumors in this vicinity may be extraordinarily elusive and difficult to discover, especially if there is a marked coloptasis, in which case this portion of the intestinal tract may be lying on the floor of the pelvis. While the hepatic flexure is ordinarily rather firmly fixed, it may nevertheless be ptosed, move with respiration, or be extremely inaccessible; the ascending colon and cecum are more amenable to palpation, but here again difficulties in the way of movability of the gut, location of the growth, thickness of the abdominal wall, and sensitiveness of the patient may retard the prompt discovery of the growth.

Tenderness always accompanies intestinal cancer, especially if infiltration has taken place into surrounding tissue or if the tumor is growing rapidly and is encountering interference in its expansion and should prove helpful in locating the new growth. Should the intestinal growth adhere to the spleen, liver, or kidney, its discovery would be more difficult; indeed, at times it is quite impossible to palpate a tumor even in the presence of all clinical signs, including complete obstruction. Because of the tendency of many intestinal tumors to be displaced under varying conditions, one may at times consider the original diagnosis incorrect; therefore it is of vast importance to examine the patient in various positions, and under different conditions, *i. e.*, deep inspiration and expiration, etc. A large fecal accumulation, dammed back of a small original growth may be mistaken for the tumor mass itself; however in the majority of cases the tumor mass is extremely hard and resistant to pressure and indentations on its surface cannot be produced; further, it is seldom ball-like but rather tends to run along the gut wall, and as a general rule it is sensitive to pressure, which sensitiveness is very exquisite if inflammation accompanies the process. One is seldom able to palpate a tumor of the small intestine, chiefly because of its movability.

A digital-rectal examination is of fundamental importance in all abdominal cases. The rectum should be thoroughly emptied by an enema, after which a close external inspection is made for ulcers, fissures, fistulæ or hemorrhoids; the finger then carefully introduced into the rectum will reveal a pathologic state, if such exists, as well as its character and proximity to the sphincter; upon withdrawal of the finger the material upon the cot should be examined for blood, mucus, pus or necrotic tissue; if the growth is soft, juicy and markedly degenerated, an ichorous and very offensive odor will be noted. If nothing be palpated by the finger, instrumental investigation should be instituted.

Percussion.—This procedure is practically devoid of value as regards determining the presence and location of an intestinal tumor.

Auscultation.—This cannot be relied upon to yield information of true diagnostic value, although rumblings, squirting sounds and the passage of gas, if confined to certain areas, may prove of value in localizing the lesion.

Stomach Contents.—There is nothing in the findings of the gastric contents which is characteristic of tumor formation in the intestine, except where intestinal contents are found with duodenal occlusion, or fecal material when an obstruction occurs further along the gut.

LABORATORY FINDINGS.—*Feces.*—A study of the feces may be extremely helpful in diagnosing tumors of the intestinal tract, especially those of a malignant nature. The various characteristics will necessarily be dependent upon the location, the extent of inflammation, catarrhal or ulcerative changes of the neoplasm and contiguous parts of the intestine.

A great deal of dependence is placed upon the continued finding of occult blood as evidence of malignancy. Should the growth be large and of the soft ulcerative type the stools may present a tarry appearance, although this finding is usually in the late stage. While copious hemorrhages in malignant tumors are exceptional, yet such a complication may be encountered; further tumor masses may slough a great deal, causing the disgusting and ichorous odor so often present.

When the lesion is in the sigmoid or rectum the stools are likely to be numerous, small and composed of mucus, pus and blood, the latter tending to be rather fresh. Should the shape of the stool be markedly changed, *i. e.*, pencil form, an obstruction in the rectum is suggested, although such a condition of the feces is often found in spastic states; again an obstruction may be present high up and the feces be formed again distal to the tumor, thus presenting a normally formed stool when evacuated; also in obstruction high up the sheep-dung, extremely hard stool occasionally appears.

On account of protein putrefaction, the reaction of the stool is usually alkaline. At times we meet with liquid diarrheal stools, in which constipated masses may be found; this combination often means irritability or inflammation of the intestine due to primary constipation. Occasionally diarrhea appears as the initial symptom of an intestinal growth; as the process develops this often alternates with constipation. While rare, yet at times fragments of the growth may be discovered in the feces.

SPECIAL EXAMINATIONS.—*Functional Tests* (*Instrumental Examinations*).—A most careful and thorough inspection of the anal region, followed by digital-rectal exploration, should always precede instrumental investigation of the rectum. It is of the utmost importance that a thorough rectal and sigmoidoscopic examination be made in every case of supposed intestinal involvement. If there be no contra-indications, a laxative is given to clear the intestinal tract thoroughly, this being followed by one or two enemas, the latter given two hours before instrumentation is applied. The patient is placed in the knee-chest position, the instrument introduced and the tube carefully directed through the lumen; any obstruction of the gut, nature of same and the degree of occlusion, the general character of the mucous membrane, whether it is inflamed, ulcerated, covered with mucus, pus or blood, or the site of polypoid or other growths should be noted. If one or more of the above conditions are encountered, tuberculosis, lues, carcinoma or parasitic invasion should be considered.

X-RAY EXAMINATION.—The administration of barium by mouth and in the form of enemata, in x-ray examinations, is of incalculable value in the diagnosis of tumors of the intestine. By this method the site of the growth and extent of involvement may be accurately determined, through filling defects and other roentgenologic signs.

Diagnosis.—CLINICAL HISTORY.—The presence of an intestinal tumor should be suspected with a history of the following: gradually increasing train of symptoms, fullness and distress, gaseous distention, later colic, interference with normal bowel evacuation, constipation, diarrhea, or alternation of both, later leading to various obstructive and toxic symptoms; anorexia, sitophobia, or possibly both; loss of weight, anemia, and general asthenia with cachexia.

PHYSICAL FINDINGS.—Our knowledge of a case may be greatly enhanced by observation, palpation and to a much less extent percussion and auscultation. The general bodily characteristics, state of nutrition, color of skin and mucous membranes, general demeanor, locally the contour of the abdomen, irregularities produced by local or general distention because of gas and growths or visible peristalsis should be observed and noted.

Palpation, while many times valueless, may nevertheless assist in localizing tenderness, frequently the earliest manifestation of tumor growth; this may be followed later by localized resistance and muscle spasm; finally a tumor may be detected and large distended coils of intestine may be palpated. Digital-rectal examination should be made in every case.

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Percussion cannot be depended upon to give accurate knowledge and is therefore practically valueless in diagnosing and localizing intestinal tumors.

Auscultation may be helpful in discovering the squirting and gurgling sounds accompanying stenosis.

SPECIAL EXAMINATIONS.—Digital-rectal exploration, instrumental sigmoidoscopic investigations and x-ray examinations are of invaluable help, and should always be instituted. Particles of tissue thus secured should be examined microscopically and the stools carefully searched for occult blood.

Complications.—Complications occasionally accompany intestinal tumors. While metastasis is rare, when it does occur, usually with a primary focus in the rectum or sigmoid, the most frequent site is the liver. A primary growth in either of these locations may be quite small, the secondary growth very large, in which event the seat of the disease may easily be overlooked.

Perforation of the intestine may occur into the stomach, and be followed by lienteric diarrhea, or into the bladder with subsequent passage of fecal material and gas through the urethra. The peritoneum may become involved, as may any other abdominal organs, because of extension of the growth; areas of circumscribed low grade peritonitis may be set up by slow but continual irritation of the tumor. Perforation with acute peritonitis rarely occurs, and skeletal and external lymph-gland involvement is unusual. Cases have been reported where attachment to the abdominal wall has occurred, finally leading to a fistulous communication with the exterior, the condition quite often being mistaken for an appendical or pelvic abscess.

Treatment.—PROPHYLAXIS.—Having no knowledge of the etiology of tumors, we cannot apply prophylactic measures. Treatment must be **symptomatic** in every particular. The patient should be placed in the very best environment and all rules of **proper hygiene** observed. The **diet** should be nutritious, palatable and of the proper consistency: if obstructive symptoms are present, first starvation, followed by liquids; then the soft, bland, nonirritating types of food leaving the minimum of residue should be given, such as watery gruels, soft strained cereals, soft eggs, cream and clear soups, purée of fruit and vegetables, and soft desserts. Crackers, zwieback and crusts of rolls may be taken if thoroughly masticated. Milk and cream may be added to the above, although these are often contra-indicated if diarrhea be present. It is more advisable to administer a small quantity of nourishment at frequent intervals than large meals some hours apart.

The symptoms should be treated as they arise; if constipation is complained of, enemas should be used and cathartics eliminated. Should diarrhea be present—provided it is not the "diarrhea of constipation"—thorough drainage by laxatives should be instituted using enemas when necessary in order to eliminate the putrid foul material often present; these measures should be followed by the usual astringents, **bismuth, tannic acid** and other like preparations, and if necessary, additions of **opium** in various forms.

Pain is best controlled by **belladonna, benzyl benzonate, bromids, veronal, codein, pantopon, Schlesinger's solution** and **morphin,** as the

occasion may demand. Local applications of heat or cold are helpful at times.

Upbuilding treatment is best accomplished by the **hypodermic use** of various combinations of **iron, arsenic, strychnin** and the **glycerophosphates. Olive oil rubs** may be used to assist a failing nutrition.

The use of the **x-ray** is considered valuable in a small percentage of cases, and lately massive and deeply penetrating doses are being applied; as yet it is too early to report on results, although in the authors' experiences this method of treatment has practically been barren of success.

Radium has likewise failed, although a few very miraculous results have been reported with the sarcomatous type of tumor.

Surgery is the only method of treatment which offers any hope, and then only when instituted before metastasis has occurred.

Prognosis.—As to Recovery.—Malignant tumors of the intestine usually result fatally because, from their location and the insidious nature of the disease, they are generally discovered only after extensive involvement renders surgery almost valueless. The prognosis in benign tumors is favorable if the growth has not become too comprehensively involved in the surrounding tissues; this, however, rarely occurs.

As to Function.—The motor secretory and sensory powers of the intestine are all greatly impaired and permanently damaged unless surgical interference, radium or x-ray proves successful.

Pathology.—The most frequent form of malignant tumor arising in the intestine is the cylindrical epithelial cell carcinoma, which has its origin in the cylindrical cells, especially of the glandular portions.

Statistics show that of all cases of carcinoma, from 8.5 to 12 per cent. attack the intestinal tract and of this number only from 2.5 to 3 per cent. involve the small intestine. About 4 per cent. of all intestinal carcinoma are found in the duodenum, and growths have been reported in all three portions of this part of the digestive tract. This condition may follow an ulcer in the first portion, causing obstruction, with subsequent dilatation of the stomach. Adhesions and metastases are usually added to the stenotic complication.

Regarding involvement of the second portion, the papilla of Vater is the usual site of election, with a resultant occlusion of the duct, jaundice and the usual gastric manifestations. Frequently the growths at this point assume a papillomatous type with marked extension and tendency to infection; further, hemorrhage is likely to occur. The histogenesis is usually that of cylindrical cell adenocarcinoma. While cases presenting the same gross and microscopic picture have been reported, carcinoma of the third portion of the duodenum is extremely rare.

Carcinomata of the ileum and jejunum are quite unusual, representing about 3 per cent. of all intestinal cancers, and occur at an earlier age than those found in the large intestine. They may be of single or multiple polypoid type, and if allowed to grow, they will show the usual clinical manifestations of intestinal stenosis, dilatation, ulceration, infection and metastatic growth.

Carcinoma of the appendix, although rare, does occur. Two varieties

have been described, the gelatinous adenocarcinoma, and the mixed alveolar carcinoma. The tip of the appendix seems to be the point of attack in the majority of cases, and often the presence of the growth is not noted unless microscopic examination is made. Cystic degeneration of the tumor, adhesions, diffuse enlargement of the organ, and projection of the mass into the cecum, with resultant constriction or obstruction have been observed. Mesenteric and retroperitoneal glands may become involved and general peritoneal carcinosis may ensue. The clinical course resembles that of chronic appendicitis. Carcinoma of the large intestine occurs most frequently in the rectum, sigmoid, and then from the cecum toward the rectum. Multiple polyposis possibly plays a rôle as regards tissue predisposition, but there as elsewhere the question of etiology is yet unsolved. Some writers claim that carcinoma may develop from hemorrhoids, others that the carcinoma precedes the hemorrhoidal state. Many authorities consider trauma a factor of importance in the production of rectal carcinoma as well as in the formation of malignant tumors at the cecum, the flexures and rectal valves.

An accepted classification of large intestine carcinomata follows:

1. Adenoma distruens.
2. Fibrocarcinoma.
3. Gelatinous adenocarcinoma.
4. Papillary carcinoma.
5. Multiple carcinoma following polyposis.
6. Squamous cell carcinoma.
7. Melanoma.

Secondary infection is an important factor in the progress of carcinoma of the large intestine, sometimes leading to suppuration, perforation, peritonitis, and fistulous communications with other organs, such as the bladder, stomach, small intestines or adjoining coils of the large bowel. Above the stricture the intestine becomes first hypertrophied, then dilated, followed by inflammation with an ensuing colitis; if the thinning process should continue, perforation may occur. Cachexia is the result of these secondary complications.

As a general rule intestinal tumors grow more slowly and metastasize to a very much less extent than carcinoma elsewhere. Hauser, Ewing, and others have shown that the disease arises from a circumscribed area of mucosa, the glands become enlarged and the lumina elongated and bifurcated; the surface epithelium grows wildly in all directions, breaks through the basement membrane, and invades the supporting stroma. Cancer cells, thus set free, invade the surrounding structures and metastasize in regional glands and distant organs. The adenoma destruens is first circumscribed but soon spreads and develops usually into a large obstructive mass and shows early ulceration. Fibrocarcinoma is a hard, circumscribed growth usually involving all the coats, in many cases resulting in a tight annular stricture. There is an abundance of connective tissue in this particular type of cancer.

Gelatinous adenocarcinoma of the large intestine is frequently encountered. This variety spreads wildly and involves quite a large area, but stenosis is not so likely to occur as in the fibrous type. Ulceration takes place early and may excavate a thick, rigid tube.

Intestinal polyposis occurs in the rectum in the majority of cases, and may spread to other portions of the intestinal tract. Supposedly produced by continued irritation it has a marked tendency to develop into malignant adenoma or carcinoma. In advanced stages hemorrhage, anemia, intussusception and prolapse may occur.

Squamous cell carcinoma may arise from the anal epidermis or by metaplasia of the lower rectal mucosa and it is very malignant. Very few cases of melanoma of the rectum have been reported.

Sarcoma is a malignant tumor composed of cells of the connective tissue type and attacks both the large and small intestine. It is, however, much rarer than carcinoma but immediate and secondary effects are practically the same.

Lymphosarcoma is a malignant neoplasm arising in lymphatic tissue from proliferation of atypical lymphocytes; it may be localized or diffuse. It is usually found in the lower ileum but may appear in any part of the intestinal tract, as a localized thickening which spreads, as do other malignant tumors, and later shows marked evidence of ulceration. This type is often found in childhood and has a decided tendency to metastasize in the early stage. Retroperitoneal lymphosarcoma, in which condition the lymph nodes are attacked by large tumor formations, is a typical lesion of this variety of tumor.

Myoma is a tumor of muscle tissue, sometimes occurring in the intestinal tract, is benign in character, and may be single or multiple. Adenoma, defined as an organoid tumor which reproduces the structure of a gland, belongs to this class of growths.

The lipoma, a tumor composed of fat tissue, is a rare inhabitant of the intestinal tract, as are myxoma (a tumor composed of mucous tissue), and fibroma (a tumor composed chiefly of connective tissue).

Other Forms of Intestinal Tumor.—Sarcoma and lymphosarcoma present the same syndrome as described for carcinoma with one or two exceptions; they are more malignant and rapidly growing types of tumor tend to attack younger people and are more likely to be found in the small intestine. Their treatment is identical with that of carcinoma. In addition to the malignant neoplasms mentioned above, there are various benign tumors, *i. e.*, lipomata, myomata, adenomata, polypi (single-multiple). Should they become sufficiently enlarged to cause complete occlusion the picture is analogous to that of malignant obstruction; except for this benign and malignant tumors of the intestine have no symptoms in common; there is a total absence of the cachexia, toxemia and concomitant symptoms so prominent in carcinomatous and sarcomatous tumors; also benign tumors do not metastasize.

CHAPTER XXXI

TUBERCULOSIS OF THE INTESTINE

BY THOMAS R. BROWN, M.D.,
and
ERNEST H. GAITHER, M.D.

Etiology, p. 773; Symptomatology, p. 774; Diagnosis, p. 776; Complications, p. 777; Treatment, p. 777; Prognosis, p. 778; Pathology, p. 778.

Tuberculosis of the intestines, tuberculous enteritis and tuberculous colitis, hyperplastic tuberculosis of the cecum and colon, tuberculosis of the rectum with fistula, primary and secondary tuberculosis of large or small bowel, tabes mesenterica (in childhood) are the terms employed to designate the various types or forms in which infection with the tubercle bacillus, human or bovine, affects the intestinal tract.

Etiology.—Since the great majority of cases represent a late intestinal infection following a primary infection of the respiratory system, all those factors which increase the incidence of pulmonary tuberculosis will *ipso facto* increase the incidence of the intestinal forms of the disease. Incidentally the very striking and steady diminution in deaths from pulmonary tuberculosis as shown by statistics of the last four years—notably in the State of New York—will probably be paralleled by a corresponding diminution of cases with intestinal involvement.

We meet with intestinal tuberculosis in various forms:

First, primary intestinal tuberculosis where no evidence of infection elsewhere is noted. The infection here, in the majority of cases, according to the more recent careful studies is due to the bovine type of the bacillus. There is an increasing prevalence of this type of the disease not only in children but also in adults in those countries where, coincident with a lowered resistance, there has been a great increase of tuberculosis among cattle. Here the infection, according to most observers, is due to the taking of milk or butter from such infected animals, or to the eating of meat from tuberculous cattle. Some, however, still contend that such primary tuberculous enterocolitis does not exist, and that the primary seat of infection is elsewhere, usually in lungs or bronchial lymph glands; but the weight of the evidence is in favor of there being a primary intestinal tuberculosis, etiologically as well as clinically. Calmette has always insisted that in the majority of cases, even those with extensive pulmonary tuberculosis and little or no obvious intestinal involvement, the primary lesion is in the intestine, a view, however, held by very few other students in this field. Certainly the fact that more than half the cases of this primary form in children show the bovine bacillus speaks strongly for infection by mouth and not by blood or lymph, borne from a primary lesion elsewhere. On the other hand it is well known that bovine bacilli as well as human bacilli may be swallowed without causing infection. Probably the acid gastric juice plays a large rôle in this connection.

Second, there is secondary intestinal enterocolitis which, in the vast majority of cases, represents a terminal picture in extensive pulmonary involvement. These obviously are due to infection with the human bacillus. The more one studies this group of cases the more one feels that in the great majority intestinal lesions occur; some figures give this incidence as high as 90 per cent.

Third, there is the hypertrophic or hyperplastic form, usually met with in the cecum, occasionally in the colon, sigmoid or rectum. According to most observers, this form is due to the human bacillus and secondary to some focus, elsewhere often very slight, usually in the lung or lymph glands, but occasionally in the urogenital apparatus. This secondary infection probably represents the action of an attenuated strain of the microörganism with hyperplasia, *not* ulceration, dominating the picture.

Fourth, there is the form which may be met with as the local manifestation of a miliary tuberculosis. Here the lesions are rarely, if ever, recognized *intra vitam.*

Fifth, one may find intestinal tuberculosis manifesting itself as an extension into the intestine of a diffuse or localized tuberculous peritonitis. This is relatively rare, though not unknown, and represents the reverse of the normal picture, that is, tuberculous enterocolitis with extension of the tuberculous process to the peritoneum.

In the case of hyperplastic tuberculosis, most commonly found in the ileocecal region we have diffuse small cell infiltration with giant cells and bacilli. Subsequently fibrous tissue forms with marked increase of thickness of the wall, and narrowing of the lumen. This is followed by involvement of the adjacent lymph glands and ulceration as a rule. In the rectum there is a striking tendency to a breaking down of the ulcer and the formation of abscess and fistula, and often of subsequent stenosis.

Symptomatology.—As tuberculous infection of the intestinal tract is met with in various forms or types, so its symptomatology varies very strikingly. In the terminal ulcerative type, the most common by far, the usual symptoms are: diarrhea in increasing intensity, melena sometimes, but more often occult blood; pain, often varying markedly in intensity, sometimes absent entirely; abdominal tenderness; and increasing loss of weight, and irregular temperature. The loose stool is usually alkaline, shows considerable undigested residue, often contains mucus, pus and blood, and usually tubercle bacilli. In cases associated with active, open pulmonary lesion, the presence of tubercle bacilli in the stools is of relatively little diagnostic value. The author has seen a few cases of extensive hemorrhage, but they are relatively rare.

in children the picture of the primary form is sometimes simply that of an intractable intestinal infection of the nontuberculous type—diarrhea, wasting, sometimes pain, irregular temperature and very occasionally sign of perforation, which are usually localized and sometimes followed by abscess formation. In this group of cases, with no open active pulmonary lesion, the finding of tubercle bacilli, usually of the bovine type, is of great diagnostic importance. In children, in certain cases, we have a striking combination of the symptoms of a tuberculous enteritis and a tuberculous peritonitis, characterized by marked involvement of the mesenteric lymph glands and liver, with the lungs almost or entirely free of infection. The glands may be so large as to be palpable, and very rarely may produce definite obstructive symptoms.

In all the above forms, pain, if present, is more likely to be constant than paroxysmal; the abdomen is usually sensitive to palpation even when the patient does not complain of pain. In most cases fever is present, and this is increased by exercise. One usually finds secondary anemia, and sometimes an oligemia due to the rapid loss of weight of the patient and the corresponding concentration of the blood. In most of the above types of intestinal tuberculosis the clinical picture usually becomes steadily worse until death. In the ulcerative type as usually seen, *i. e.*, the type found in the late stages of an inactive pulmonary lesion with the appearance of marked intestinal symptoms and signs, the downward course of the patient is markedly accelerated.

In the case of the hypertrophic ileocecal tuberculosis, on the other hand, the picture is quite different; the mechanical factors, not the ulcerative and toxic, dominate the picture. There is a gradually increasing discomfort with the usual dyspeptic symptoms—fullness, loss of appetite, constipation, sometimes alternating with rather violent diarrheal outbreaks; later there are present signs of obstruction, colicky pains, distention of proximal gut, loss of weight, sometimes visible peristalsis, or, in other words, the usual symptoms of intestinal stenosis. Occasionally, in the later stages, profuse hemorrhages occur; for in this stage ulceration, sometimes quite extensive, is manifest. A tumor mass is made out, not quite so hard or nodular as a malignant neoplasm though frequently mistaken for it. Fever is sometimes present, sometimes absent. The stool usually shows the foodstuffs well digested; frequently mucus is found. The finding of the tubercle bacilli is of paramount importance as this form of intestinal tuberculosis is very often associated with inactive and apparently healed lesions in the lungs. Much less frequently a similar condition may be found in the colon or in the sigmoid.

In certain cases the rectum seems to be the seat of election of the tuberculous process; if the ulceration predominates, the stools are more frequent; pus, blood and mucus are more likely to be found microscopically; tenesmus is frequent. Fistula and perirectal ulcers are not uncommon; stenosis and partial obstruction are frequently seen. In certain cases of tuberculous abscess of the intestine, intestinal obstruction or stricture may occur. Sometimes the appendix and the periappendicular tissues are the parts of the tract most involved, and the picture may simulate almost exactly that of chronic appendicitis, but very rarely that of acute appendicitis.

Diagnosis.—In the vast majority of cases, the diagnosis is made only from a very careful consideration of the history of general, as well as of abdominal symptoms, and often is made definite only by certain additional rectal, sigmoidoscopic, stool and intensive x-ray studies.

In the case of adults and older children tuberculin tests or complement fixation tests are of relatively little value; because of the frequency of the association of intestinal with pulmonary tuberculosis, and of the incidence of positive tuberculin reactions in many apparently absolutely healthy individuals.

In the most common type, ulcerative enterocolitis associated with primary pulmonary tuberculosis, the question is whether the intestine is or is not involved. More rapid loss of weight, more irregular temperatures, with loss of appetite, and slight gastric and intestinal symptoms so common in any active tuberculous lesion, or more or less severe abdominal pain, and frequently increasing diarrhea, instead of the vague digestive symptoms, speak strongly in favor of an intestinal involvement which, as we have said before, is present in the vast majority, if not in all cases in the final stages of pulmonary tuberculosis. Certainly intestinal involvement is of truly grave prognosis in pulmonary tuberculosis. Primary tuberculous enterocolitis in infants and young children should always be thought of in cases of unusual symptomatology, or when the usual therapy for nontuberculous infections yields little or no relief. Here the discovery of the bacillus, usually of the bovine type, in the stool is of great significance; while obviously in the first group the finding of bacilli in the stool is of relatively no importance.

The possibility of a tuberculous peritonitis should always be kept in mind in cases of typhoid fever with unusual symptoms, especially those of peculiarly protracted course. Also it must not be overlooked that a chronic, subacute or even acute appendicitis may occasionally be due to a tuberculous infection. In either form of appendicitis leukopenia and the presence of the tubercle bacilli in the stool are very significant.

In the hyperplastic or hypertrophic form with palpable tumor the case is usually at first regarded either as one of chronic inflammation due to the ordinary pyogenic organisms or as one of malignant disease. Very careful x-ray studies showing filling defect, spasm and hypermotility (Brown and Sampson) are very helpful if associated with definite, although very often slight or even arrested tuberculosis elsewhere. And as Carman well says "a lesion roentgenologically demonstrated in the ileocecal coil with irregularity in the bowel picture, although it may represent only ulcerative lesion is probably tuberculous if pulmonary tuberculosis is present." In this group of cases the finding of the bacilli in the stool is of great significance.

In rectal tuberculosis the character of the ulceration or of the stricture, the demonstration of a fistula or a perirectal abscess, and the presence of the bacilli are the most important diagnostic points. Proctosigmoidoscopy is invaluable.

Complications.—In the ulcerative form secondary to extensive tuberculosis elsewhere, small hemorrhages are common, and extensive hemorrhages rare; partial obstruction is sometimes met with; while perforation occurs very occasionally with the development of local peritonitis, and very rarely, with general peritonitis, but sometimes with a local tuberculous or pyogenic abscess.

In the hypertrophic form obstructive attacks are common and very extensive hemorrhages occasional.

In the rectal form stricture, fistula in ano and perirectal abscess are rather frequent, while rectovesical or rectovaginal fistulæ are not so common.

In the vast majority of cases varying degrees of obstruction are met with as sequelæ.

Treatment.—As in every other form of tuberculosis, **prophylaxis** is of far more significance than treatment. The value of such preventive therapy, better living and housing conditions, improved nutrition, careful supervision of cattle and milk, isolation of or the inculcation of proper habits in those with active lesions, proper methods of cleansing or disinfecting foci or potential foci, pasteurization of milk, tuberculosis classes with their invaluable benefit to the well and the sick, etc., is shown in the very striking reduction in tuberculosis incidence and tuberculosis mortality within recent years.

In cases with pulmonary, buccal or laryngeal tuberculosis, the patient should be especially **cautioned against swallowing the sputum.** While the hydrochloric acid of the stomach is some protection, it is not an absolute defense against infection of stomach or intestines, and in addition, in many cases, especially in the later stages of pulmonary infection, gastric subacidity or anacidity is frequently met with, and the bacilli reach the intestines unchanged and thrive in the alkaline medium thereof.

With regard to special treatment in the ulcerative forms of intestinal tuberculosis the usual regulations as to **rest, local heat,** and a **nonirritating dietary** should be insisted upon. If the diarrhea is intractable, **ileostomy** or **cecostomy** has given some temporary relief occasionally. In intestinal tuberculosis specific treatment, *i. e.*, treatment with some form of tuberculin has been singularly disappointing as have the myriad forms of chemotherapy which have been suggested. Included in the latter are the treatments with sugar, various organic preparations of copper, iodin, sulphur dioxid, various salts of sodium, magnesium, calcium or potassium, or of certain of the rarer metals such as neodyme. Preventive vaccination has been suggested by some; the authors have had no experience with it. Certainly in most cases of ulcerative enterocolitis in addition to general hygienic measures, the only treatment of value is that designed to lessen the diarrhea and to allay the pain. Rest, soft nonirritating food, local heat, occasionally colonic irrigations, and sometimes sedatives (opiates usually being required) are indicated, but are purely palliative. Many of these cases have been treated by x-rays, the ultra violet rays, and a few with radium, while quite recently very striking success in the treatment of intestinal tuberculosis has been claimed for the quartz lamp.

Surgical treatment consisting of **resection of the involved area** has given really brilliant results and sometimes apparent cures in certain of the cases of localized hypertrophic tuberculosis. In the diffuse ulcerative forms, however, surgery has obviously been rarely tried, although a few cases seem to have been temporarily helped by appendicostomy, ileostomy or cecostomy with subsequent irrigation. There are probably a small number of cases of ulcerative enterocolitis of tuberculous origin that heal; most of these are probably cases with little or no clinical manifestations, or those with slight intestinal symptoms or very little involvement elsewhere; but the appearance of definite signs and symptoms of intestinal involvement in persons with pulmonary tuberculosis is of evil omen.

Prognosis.—Concerning treatment and prognosis we certainly must feel that at the present time our greatest hope lies in preventive medicine, increased individual resistance and the furthering of all means that lessen the chance of massive infection.

Pathology.—With regard to pathological changes, it is well to remember that the tubercle bacilli choose the lymphatic follicles as their seat of special predilection. These subsequently undergo caseation and thus the ulcer develops, but not so extensively as in typhoid. The lower portion of the ileum is most affected, either alone or associated with similar lesions in the cecum and ascending colon, and less often with lesions in the duodenum, jejunum, descending colon and rectum (Schmidt). Further ulcers may develop in this region and tend to lead out in a transverse direction following the course of the blood-vessels, and affect not only the mucosa and muscularis, but even in some cases the circular and longitudinal muscles, with subsequent contraction; in which case a ringlike stenosis of greater or less extent, and not infrequently a local peritonitis may develop from extension to the peritoneal coat and subsequent adhesion formation. Rarely perforation occurs, and not infrequently tuberculosis of the mesenteric glands; sometimes healed scars are present.

CHAPTER XXXII

SYPHILIS OF THE INTESTINES

By Thomas R. Brown, M.D.,
and
Ernest H. Gaither, M.D.

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Symptomatology, p. 780:—Laboratory findings, p. 781; Special examinations, p. 782.
Diagnosis, p. 782.
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Clinical varieties and types, p. 782.
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Pathological anatomy, p. 782.

Etiology.—Until recently luetic lesions of the intestine, with the exception of the rare rectal chancre usually due to coitus per anum, were rarely, if ever, diagnosticated *intra vitam*, and were therefore more interesting anatomically than clinically. However, since the discovery of treponema pallidum and salvarsan, the latter furnishing us with a means of applying the therapeutic test more satisfactorily than either mercury or the iodids, clinicians have begun to realize that intestinal lues, while rare, is more common than hitherto supposed; and like syphilis of the stomach, an increasing number of cases is being diagnosed during life and treated appropriately. It may be met with as a primary lesion; the secondary stage may have definite intestinal manifestations, but it usually represents a tertiary lesion of the disease. It may also be a manifestation of congenital lues. It is also highly probable that in certain definite pathological conditions of the intestines—some at least with clinical manifestations, notably arteriosclerosis and amyloid disease—syphilis plays a definite etiological rôle in a number of cases, while certain of the paraluetic lesions, such as tabes dorsalis, are sometimes associated with striking intestinal manifestations, notably colonic crises and intractable diarrhea.

Why certain organs should be the organs of election for the major manifestations of the disease, and why in certain cases—relatively very few—the intestines should be involved are questions which cannot be answered definitely.

Contrary to the authors' experience, many observers state that females are more likely to be affected with acquired intestinal lues, especially in the rectum. According to Schmidt this is possibly due to the proximity of the vagina and the rectum, the frequency of the chancre, secondary lesions, and condylomata in that locality, and the ease with which such infectious secretions with the causative organisms invade the lower bowel lying near by.

The congenital form obviously is most likely to occur in childhood, often in early infancy. In acquired syphilis, the intestinal lesions are much more usual as a tertiary lesion, and are late manifestations of the disease; they are therefore somewhat more likely to be met with in middle or later life.

TRAUMA.—It is interesting to note in regard to the possible rôle of trauma, that intestinal lues is most likely to occur in that portion of the intestine which is most subject to trauma, *i. e.*, the colon, terminal ileum and rectum.

Practically all of the cases represent previous infection with the spirocheta pallida and usually give a history of a primary genital chancre followed more or less frequently by the various secondary manifestations. However, in a limited number of cases, it is quite impossible to obtain a history of primary or secondary lesions, and the diagnosis is made only by serological studies and the therapeutic test.

HEREDITARY TRANSMISSION.—Syphilis in the small intestine is rare, which, as has been noted by many, presents a relative immunity toward luetic infection. Nevertheless, it must be recognized that in hereditary syphilis some intestinal involvement, notably intestinal ulceration with diffuse infiltration of the intestinal wall sometimes associated with gummata and often with extensive periarteritis, is not uncommonly found.

TYPES OF INFECTION.—The subject is relatively too new to permit definite statements about various types. As far as can be seen a certain small proportion of cases of syphilis show intestinal involvement, but it is quite impossible to determine why infection occurs in this locale, and why the great majority show no such involvement, for there is absolutely nothing in the past history, mode of onset, etc., which would allow us to differentiate these types of cases.

Symptomatology.—There are no characteristic clinical symptoms, and it is highly probable that a considerable number of cases of intestinal syphilis are not recognized. In hereditary syphilis diarrhea (often of the dysenteriform type) without apparent cause should make one suspicious of intestinal lues, though it is often clinically impossible to differentiate it from intestinal tuberculosis or other intestinal infections.

In the case of acquired syphilis lesions of the small intestine are relatively rare. Here the symptoms are partly those due to intestinal inflammation, notably diarrhea, fever, etc., although sometimes certain of the symptoms must be regarded as being due to a partial obstruction most likely found in the terminal ileum. Thus the symptom complex may in some cases resemble an enteritis, and in other cases a small intestinal obstruction.

In the colon luetic infections are more likely to be met with and recognized than in the small intestine. Here, again, the usual symptom is rebellious diarrhea in the early stages, sometimes with dysenteriform symptoms, tenesmus, mucus, blood and pus in the stool, and sometimes fairly profuse hemorrhages. In the later stages where stricture occurs, there may be marked and occasionally intractable constipation. Now and then alternating constipation and diarrhea and at times true obstructive symptoms are the striking features of the case.

In certain cases, however, in which a definite lesion cannot be demonstrated, it is possible that the intestinal symptoms presented—diarrhea, excessive fermentation, and various manifestations of abnormal intestinal function—are to be ascribed to the associated arteriosclerosis and amyloid degeneration of luetic origin.

The symptoms due to stricture are peculiarly likely to be met with in involvement of the rectum where, secondary to caseation of the gumma, stricture frequently supervenes; although here again certain authors believe that the stricture really represents the results of a secondary infection.

In many cases it is quite impossible to recognize the symptoms as luetic in origin, as often there is no characteristic difference between this and other forms of enteritis, colitis or proctitis, although we cannot help but feel that one should become peculiarly suspicious if stenosis occurs. Perforation has been described. Very little can be made out by the ordinary methods of examination, and our knowledge must come in the main, first, from a history of previous luetic infection, and second, from a very careful investigation of the intestine by special methods. Such investigations should include a study of the stools, digital and sigmoidoscopic examination of distal bowel, and careful roentgenological studies, using both fluoroscopic and radiographic methods, with barium both by mouth and by enema. A stricture of the rectum perceptible to the finger should make us extremely suspicious of lues. Looseness of the stools associated with the finding of blood, pus and mucus in the early stages, and the later appearance of pencil or ribbon stools accompanied by a feeling of obstruction, local discomfort and insufficient defecation should further direct our attention to a possible luetic state. In the x-ray picture ulceration, stenosis or obstruction is observed, although there is nothing especially characteristic about this, and the finding is usually regarded as suggesting malignancy. When the obstruction is marked, one can get characteristic signs from the distention in the proximal portion of the gut. It should not be forgotten that symptoms of extreme colonic pain, so-called colonic crises or, a motor equivalent, an intractable diarrhea, may be met with in the paraluetic infection—tabes dorsalis—without demonstrable pathologic changes; in all probability it is due to secondary disturbances or to disease of the nervous mechanism of the intestine.

Laboratory Findings.—Obviously, the most essential laboratory finding is the demonstration by serological methods of a positive reaction in the blood (or in the spinal fluid if cerebrospinal syphilis is suspected); for although we realize that in a few cases syphilis may be present with a negative reaction, certainly it is almost impossible to diagnosticate intestinal lues without such a reaction, or without a definite history of primary luetic infection. The spirochetæ have occasionally been demonstrated in the secretions from a luetic intestinal infection. The stool, as stated before, often gives us valuable information: first, blood, pus, mucus and looseness of the stool; later, the characteristic picture of obstruction in a pencil or ribbon stool.

Special Examinations.—It is needless to say that a very careful proctoscopic and sigmoidoscopic examination should be made in every case, although it is extremely difficult to differentiate by this method the ulcerative lesions of syphilis from an ulcerating neoplasm or ulcerating colitis or sigmoiditis of nonspecific origin, or a tuberculous ulcerative lesion. Some feel that there are certain peculiarities of the luetic lesion, such as more general distribution and more extensive and less deep ulceration. These, to a certain extent, show more definitely in the x-ray findings. Greater irregularity, deeper ulceration and a sharper limitation between the lesion and the normal intestine have been described as being more characteristic of carcinoma, while the reverse speaks more in favor of lues.

Diagnosis.—It is beyond question that most cases of intestinal lues are not diagnosticated. In congenital lues the diarrhea is often considered as a simple enteritis, enterocolitis or a tuberculosis-lesion, while the same holds good for acquired syphilis where, in addition, unquestionably a certain proportion of the cases are incorrectly diagnosed as malignant disease. In the secondary stage, the diarrhea may be due to the disease itself, whereas the picture presented is sometimes that of a true typhoid state associated with diarrhea, although in the secondary stage in many cases it is highly probable that the diarrhea is due not so much to the disease itself as to the therapy employed. In reaching a diagnosis nothing is more important than a carefully taken history in which there are definite or, at least, suggestive symptoms of a primary infection. The infection may manifest itself in a positive reaction in the blood and also in the spinal fluid, Argyll Robertson pupil, absent knee jerks in the case of cerebrospinal lues, and the history of diarrhea followed by symptoms of obstruction, or it may be suggested by the stools, roentgenologic studies, and digital-rectal and proctosigmoidoscopic examinations. In certain cases, the therapeutic test may aid us greatly in making our diagnosis. In many instances, however, the case history and luetic manifestations elsewhere are the most important factors in making a provisional diagnosis, which sometimes is made only more probable, if not certain, by the exclusion of other causes.

Complications.—As mentioned before, the one most important complication is partial obstruction. In certain cases extensive hemorrhage occurs; perforation is found with extreme rarity. Secondary infection is common in all intestinal lesions whatever the primary cause, and, as said before, it is regarded by some as the fundamental cause of the stricture met with in such relative frequency in this disease. As in all other forms of lues, especially the untreated or incompletely treated cases, marked nervous manifestations, anemia and fever with no apparent cause are sometimes met with. The most important sequela is permanent stricture, most frequently seen in the large intestine, but not unknown in the small bowel, particularly in the terminal ileum, and very occasionally in the jejunum.

Association with Other Diseases.—It must not be forgotten that it is quite possible to have syphilis and other diseases present in the same individual, and, in certain cases, it will be quite impossible to determine whether the intestinal lesion is luetic, malignant, or due to some other cause, until after specific therapy has been tried or other means employed to determine the nature of the lesion.

Clinical Varieties and Types.—The disease is not sufficiently known nor has it been studied thoroughly enough to allow us to speak of types. Perhaps the simplest way would be to differentiate the intestinal manifestations of the disease as to the place of special involvement,—small intestines, colon, sigmoid or rectum; or to differentiate them on clinical grounds—cases where irritative phenomena, notably diarrhea, and ulceration dominate the picture, or cases with obstructive phenomena; however, we sometimes get an alternation of both groups of the above cases.

Treatment.—The treatment of the disease is fundamentally antisyphilitic, and includes such therapeutics as **salvarsan, iodids** and **mercury**. The degree of success of **serological treatment** with regard to intestinal function is obviously dependent upon how early such treatment is instituted and upon the degree of involvement of the intestine. Superficial ulceration or gummata which have not undergone extensive caseation with subsequent contraction may obviously be materially benefited and possibly entirely cured clinically by specific antisyphilitic treatment. On the other hand, if stricture has taken place the benefit will be relatively much less, and it will be necessary to determine whether the patient can be made comfortable by appropriate dietetic and medicinal means, or whether surgical treatment—ileostomy, colostomy, or even resection—is justifiable. If the obstruction is very marked, **surgical interference** will be necessary; if it is not so marked and is located in the rectum or lower sigmoid, **mechanical dilatation** may be employed. If ulceration is present, considerable comfort can sometimes be obtained by **astringent** or **antiseptic irrigations**. If diarrhea is present, the usual bland, nonirritating dietary should be employed; possibly it will be necessary to give a **liquid diet** for a short period of time, in conjunction with **bismuth** or **bolus alba**; **rest** until the diarrhea is checked must be insisted on. If pain is present, **local application of moist heat**, antispasmodics such as **belladonna**, and **opium**, or other forms of sedatives may have to be employed.

Prognosis.—The prognosis is entirely dependent upon the extent of the involvement of the intestine and the stage at which treatment is instituted. If such treatment is started before deep ulceration, extensive caseation or marked cicatrization has taken place, and is conducted over a sufficient period of time, the prognosis, at least so far as the intestinal symptoms are concerned, is good. On the other hand, if extensive and deep changes have occurred, it is highly probable that a certain degree of stenosis will be left behind. If the intestinal symptoms represent manifestations of intestinal arteriosclerosis or amyloid disease of intestinal origin, specific treatment will play a relatively small rôle. In arteriosclerosis iodids may help; but if the intestinal symptoms are due to cerebrospinal syphilis, the authors' experience leads them to believe that the prognosis is not good, even though intensive intraspinous treatment is carried out.

Pathological Anatomy.—In the primary stage, the extragenital chancre shows no essential difference from the genital. In the secondary stage, the follicles are peculiarly likely to be affected, and in a few cases the spirochetes have been demonstrated in the affected areas. In the usual or tertiary stage, ulceration is more frequent in the terminal ileum and in certain portions of the colon, but it is not unknown in the jejunum or even in the duodenum. These ulcerations have a tendency to extend circularly, are usually superficial and are associated with small cell infiltration and the vascular changes, arteritis and periarteritis. The follicles are frequently the seats of especial election of the ulcerations. In addition, gummata are found which break down and form deep ulcerations, though this probably represents a partial manifestation of secondary infection. The process is sometimes localized in the rectum, and here secondary contraction and stricture are peculiarly likely to be met with, although such strictures have also been found in the sigmoid, colon and even in the small bowel.

CHAPTER XXXIII

INTESTINAL NEUROSES

By Franklin W. White, M.D.

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GENERAL CONSIDERATIONS

Intestinal neuroses show a disturbance of the normal function of the bowel without any organic change. In these cases we have general nervous symptoms in addition to the intestinal disturbance. The more carefully such cases are studied, the fewer pure neuroses will be found; that is, many so-called neuroses of the intestines will be found to have a definite organic basis. These neuroses are not to be regarded as definite diseases but as syndromes, dependent upon one or more causes, not always clearly defined. Some are scarcely more than symptoms. Some are common, such as intestinal atony and spasm. Nervous diarrhea is quite frequently seen. Others, like pure enteralgia, intestinal fermentative dyspepsia and peristaltic unrest are rarely met with. Most of the gastro-intestinal neuroses are found more frequently in women, especially in youth and early middle age.

The muscular wall of the intestine, with the exception of that of the rectum and the secreting glands are controlled by the even balance of stimuli coming to the bowels over two pathways, the vagus and the sympathetic nerves. When this even balance is upset, any one or more of the functional disturbances may result, and we may expect many of these disturbances on account of the complexity of this nervous control and its close relation to the endocrine system. There are a few well-defined neuroses, disturbances of motion, secretion, and sensation, but less than we might expect.

Etiology.—The underlying *causes* which upset the properly balanced action of the vagus and sympathetic systems are varied; emotional states such as fatigue, worry, excitement or fear; functional neuroses such as neurasthenia and hysteria; organic visceral disease such as syphilis of the central nervous system; disturbances of metabolism and of the endocrine glands; the chronic toxemia of nephritis or tuberculosis; and toxemias arising from probably remote and perhaps concealed infections of the sinuses, tonsils, teeth, and gall-bladder.

Eppinger and Hess have called the result of vagus or sympathetic irritation vagotonus or sympatheticotonus respectively. In vagotonus there is increased tone or spasm in the intestine; spastic constipation, and increase of intestinal peristalsis: peristaltic unrest or nervous diarrhea; and spasm of the sphincters; also increased secretion of the intestinal, hepatic and other glands; and possibly also increased or abnormal sensation. In sympatheticotonus, there are atony and dilatation of the bowel, weakened peristalsis with slow pushing forward of the intestinal contents, relaxed sphincters, low secretion and lessened sensibility.

Symptoms of a general nervous condition are usually found, such as fatigue, headache, insomnia, introspection, and depression. The digestive attacks appear without clear cause, and are often very irregular, with intervals of well-being which are striking and unexpected. The attacks are much less dependent upon food than upon fatigue, mental strain, or excitement. The gastric secretion and motility are usually normal.

Diagnosis.—The well ordered function of the bowel depends upon this properly balanced action of the vagus and sympathetic system. It is important to find the causes which upset this harmonious action, which can be done only by a thorough examination of the patient. Great care is necessary in making this diagnosis of an intestinal neurosis; it should be made only when every sign of an organic intestinal lesion is absent and the condition gets better or worse, chiefly from emotional causes. Neuropathic persons with increased reflex irritability react much more actively to small pathological causes than those with a well-balanced nervous system.

The diagnosis of nervous diarrhea is often made in error. It is quite rare. Such cases are usually combinations of neurasthenia and catarrh of the bowel. In ''mucous colitis'' the outpouring of mucus may be a nervous secretory sign or the result of actual infection. Atonic constipation may be dietary as well as due to lack of nervous control of the colon. Peristaltic unrest of the small bowel may be due either to irritability of the sympathetic system or to inflammation of the mucous membrane. Neurasthenic persons with ptosis have many disagreeable symptoms, such as flatulence, abdominal pain and distress, which can be easily proved by examination of the stools to be due to an intestinal catarrh in combination with the nervous condition.

In some conditions, as spasm of the anus or intestinal atony, the diagnosis can be safely made by the busy practitioner after a routine examination; in other conditions, such as enteralgia or peristaltic unrest, it is extremely important to take the patient seriously and rule out important organic causes of abdominal pain or obstructive causes of visible intestinal peristalsis before resting on the diagnosis of a pure intestinal neurosis.

Treatment.—The patients are easily influenced for good or ill by the personality of the physician. A thorough physical examination often helps greatly by ruling out organic disease and banishing fear from the patient's mind. Of the greatest importance are **general hygienic measures**, such as a vacation, the "rest cure," diversion, occupation therapy, massage, gentle exercise, electricity, hydrotherapy and psychotherapy. It is important to avoid fatigue. Specific directions about the amount of rest, exercise, food and sleep should be given. Gain in weight, **by forced feeding**, is often useful. Simple general diets are the best without so much restriction as to interfere with appetite or nutrition. Stimulants, tea, coffee, and tobacco are to be avoided. Drugs are of minor importance except in acute attacks. **Belladonna, atropin, benzyl succinate**; hypnotics such as the **bromids, barbital** or **trional**; and simple tonics such as the **bitters, iron** and **arsenic** are most useful.

Prognosis.—This depends more upon the general condition of the patient and the stability of the nervous system than upon any local signs. Relapse and recurrence are frequent in persons who are physically inferior.

SPASM OF THE INTESTINE

In nervous persons we have irregular contraction or increased peristalsis of the bowel, or parts of it, causing in the acute cases, pain, gurgling, defecation and tenesmus, and in the chronic cases, spastic constipation.

Etiology.—Spasm is really a symptom, not a disease, though it may dominate the clinical picture in some nervous persons for a time. Except for spastic constipation, spasm is rare as a pure neurosis, that is, due purely to an increased irritability of the autonomic nervous system. On the other hand, reflex spasm of the bowel, or one portion of it, is common as a symptom of a large number of organic diseases without or within the bowel, such as diseases of the gall-bladder or appendix, catarrh or inflammation of the bowel, piles, fissure ani, lead poisoning, tabes, etc. It is needless to say that such causes provoke intestinal spasm more easily in neuropaths than in others.

Symptomatology.—The patient complains of a feeling of constriction in the abdomen or colicky pain with gurgling of gas, and perhaps passage of flatus or feces. The colon, especially the transverse colon, or sigmoid may be felt as a firmly contracted tender cord.

In mild chronic cases, we have spastic constipation as the chief symptom. Moderate grades of intestinal spasm are common, and affect certain parts of the bowel far more than others, *e. g.*, the first portion of the duodenum, transverse and descending colon, and sigmoid and sphincter ani. It is much less common in the jejunum, ileum and cecum. Very rarely, an extreme spasm may lead to intestinal obstruction, the so-called spastic ileus.

The x-ray shows some spasm of the transverse and descending colon in most constipated persons, which is often associated with atony and distention of the cecum. Less commonly there is overactive peristalsis of the bowel and the picture resembles peristaltic unrest or nervous diarrhea.

In lead poisoning, the tonic contraction involves the whole bowel but especially the small intestine and may last for a whole day. Spasm of the first portion of the duodenum is frequently seen with the x-ray as a reflex result of disease in the stomach, gall-bladder, and appendix.

It is possible that spasm of the ileocecal sphincter may occasionally cause delay in emptying the ileum with fermentation of its contents and toxic absorption; but ileal stasis is far more likely to be due to atony of the ileum or to obstructive adhesions in the ileocecal region.

Spasm of the sphincter ani is almost always due to local causes, such as piles, fissure or ulcer. It may rarely be due to pure vagotonus in a neuropathic person. It may be very painful and interfere with defecation.

Diagnosis.—The diagnosis of a pure nervous enterospasm is made from the neurotic character of the patient, the obvious local signs of spasm, and the absence of an organic reflex cause. The x-ray often discovers the spastic state of the colon or duodenum.

Treatment.—The general treatment of the functional type is the treatment of neurasthenia, with the addition of **hot applications** to the abdomen, **hoth baths** and **mineral oil** by mouth or rectum. A tablet of **extract of belladonna,** 1/4 grain (.0162 gram), or of *atropin,* 1/150 grain (.00195 gram), or of **benzyl succinate,** 5 grains (.324 gram), may be given three or four times a day, or a suppository of **extract of belladonna,** 1/2 grain (.0324 gram), **extract of hyoscyamin,** 1 grain (.065 gram), and **extract of stramonium,** 1/2 grain (.0324 gram). In painful spasm of the anus, this suppository, or one containing **cocain hydrochlorid,** 1/2 grain (.0324 gram), or **extract of opium,** 1 grain (.065 gram), may be used, or the **sphincter forcibly dilated** as for fissure.

Spasm at the rectosigmoid juncture may be treated by applications of a **saturated solution of magnesium sulphate** on **cotton sticks** made through a rectoscope as advised by Soper. The treatment of spastic constipation will be detailed elsewhere.

PERISTALTIC UNREST

(Tormina intestinorum nervosa)

This condition, first described by Kussmaul in 1878, is rare and is found chiefly in nervous persons. It is characterized by excessive peristalsis of the small intestine, which may be both visible and palpable, accompanied by abdominal discomfort and gurgling.

Etiology.—It is undoubtedly a result of increased irritability of the autonomic nervous system, which governs the peristalsis of the bowel.

Symptomatology.—The symptoms consist of a feeling of abdominal tension with rolling movements in the bowel, and rumbling and gurgling which usually are easily heard by others. There is little or no pain. The active peristalsis is often easily seen and palpated by the physician through a thin abdominal wall. It is rarely seen by the patient. Boas has described a case in which the contour of the abdomen resembled a sack of potatoes. The peristalsis travels rapidly from one portion of the small intestine to the other, stopping at the ileocecal valve. Emptying of the bowel is rare, showing limitation of the peristalsis to the small intestine. It appears at irregular intervals, lasts from a few minutes to hours, may occur at night and may be provoked by emotional causes, catamenia, or food.

Diagnosis.—The diagnosis is easy as a rule. Peristaltic unrest is rare. It occurs almost exclusively in neuropathic persons. The attacks are usually brought on by emotional causes. Digestion and defecation are apparently normal. It should be remembered that normal peristalsis of the small intestine can often be seen and felt in very thin or elderly persons. This has nothing to do with tormina, neither has the occasional rumbling of gas in the bowel heard in simple gaseous fermentation. When visible peristalsis is seen in the bowel, the early stage of intestinal obstruction must be considered and a verdict withheld until a thorough examination can be made. In intestinal obstruction there are colicky pain, vomiting and constipation; the condition is far more serious and violent, and other physical signs of obstruction are found, such as tympanites, tumor and changes in the pulse and facies.

Treatment.—The treatment is directed toward improving the tone of the nervous system and reducing vagotonus. **General hygiene**, a **rest cure**, **massage** and **hydrotherapy** are important. The most useful drugs are **bromids** in 10 grain (.65 gram) doses or **sulphate of atropin**, grains 1/150 (.00195 gram), given two or three times a day, perhaps combined with heavy **oxid of magnesia**. **Rest in bed** and **hot applications** to the abdomen may be used in trying attacks or at night. For the distress and tension, **acetphenetidin** and **acetylsalicylic acid** are useful. Codein and opium are rarely needed.

Prognosis.—The prognosis depends upon the general or nervous condition of the patient. Peristaltic unrest is disagreeable but never dangerous.

NERVOUS DIARRHEA

A diarrhea occurring abruptly and irregularly without discoverable organic cause is called nervous diarrhea. It is chiefly found in neuropathic persons but may occur in any one after emotional shock or stress.

Etiology.—There is no doubt about the existence of a purely nervous diarrhea, but the number of cases formerly so diagnosed has been greatly reduced by modern functional tests of the digestive tract together with careful examination of the feces. The typical attacks come on suddenly after some emotional stress as grief, worry, mental strain or excitement. It is a mixed neurosis, both motor and secretory. The intestinal hypermotility is similar to peristaltic unrest with the addition that the peristalsis is continued into the colon. There is also a hypersecretion of intestinal fluids resulting in large watery stools.

Symptomatology.—An explosive diarrhea is the chief symptom. The entire contents of the bowel may be emptied in watery stools within an hour. The evacuation is usually entirely painless, occasionally accompanied by ordinary abdominal colic and soreness. The oncoming of the diarrhea has no relation to the ingestion of food or drink, but is in close relation to the emotional shock. It is very variable and intermittent. It may be limited to a few movements or last for days. The patient usually has no digestive trouble between attacks.

The stools are rather odorless and loose like the normal contents of the small intestine or perhaps more watery from excessive secretion. There is no mucus, and the fat, starch, and meat residue is present in the normal proportions, though usually less digested than normal because of their rapid passage through the bowel. The microscope shows no increase of connective tissue such as is seen in the diarrhea of gastric achylia. The x-ray shows nothing except hypermotility of the bowel.

Diagnosis.—The clinical features, the close relation of the diarrhea to emotional upsets on repeated occasions with no relation to food, the association with neurasthenia, and the character of the stools just described make the diagnosis simple in many cases. In other cases we must be very careful not to overlook organic or inflammatory causes of diarrhea and a complete examination and prolonged observation are needed. We must rule out acute infections, intestinal catarrh and mucous colitis by the absence of mucus from the stools. We must look for hyperthyroidism. Minor causes of diarrhea may be overlooked, such as slight dietary errors, brief intestinal catarrhs or a few diverticulæ of the colon, and the case may be incorrectly diagnosed nervous diarrhea. It resembles the diarrhea of gastric achylia with which it is closely related, but there is a more definite emotional cause and connective tissue is absent from the stools.

The neuropath is more easily upset than a normal person by slight causes of all kinds, and some diarrheas are partly nervous and partly inflammatory or organic. The chronic and frequently recurrent diarrheas are almost never purely nervous.

Treatment.—The immediate treatment is that of any simple diarrhea. The acute attacks usually subside quickly but may easily recur. Theoretically diet is not causal and not very important; practically while the attack lasts it is better to **restrict the diet** to milk, eggs, toast, thickened soups, cereals, rice, lean meat, crackers, macaroni, custard and the like. This restriction must be brief, and a monotonous diet with malnutrition must be especially avoided. No preliminary purge is needed; general and local **sedatives** should be given at once, **tincture of belladonna**, 10 drops, or **bromids**, 10 or 15 grains (.65 or .972 gram), in a glass of water several times a day. A single dose of 10 drops of **tincture of opium** is very effective at the start of an attack if no habit formation is feared. It is very important to reassure the patient, to point out the absence of any serious disease, to caution against watching the stools too closely, and to insist that the diarrhea is really not more important than any other nervous symptom, such as nervous headache or fatigue. General treatment of the underlying nervous condition must be carried out.

Prognosis.—Nervous diarrhea is disagreeable but not serious. There may be some loss of weight, but it is often seen in plump persons. Brief recurrences may appear for years in spite of treatment. Any serious or disabling diarrhea makes one suspicious of an organic cause.

ATONY AND PARESIS OF THE BOWEL

In atony of the bowel the muscular walls have lost their tone, and do not push along their contents in a normal way, causing in the chronic form, constipation, with undue putrefaction and fermentation of the intestinal contents, and in the acute form, paresis or paralytic ileus. This is an example of sympatheticotonus.

Etiology.—Atony and paresis are really symptoms or signs, not independent diseases. The chronic form is found in fatigue, old age, the toxemia of chronic infectious diseases, also in neurasthenic, enteroptotic, and sedentary persons. It is difficult to see why spasm of the intestine (vagotonus) predominates in one person, and atony (sympatheticotonus) in another of similar make-up. The acute form follows laparotomy, shock, trauma and peritonitis.

Symptomatology.—The chronic common type is often characterized by its most prominent symptom, constipation, and will also be described under atonic constipation. Excessive putrefaction and fermentation of the intestinal contents often occurs and may lead to secondary catarrh of the bowel. It is common to assume that absorption of toxic substances from the bowel, the so-called "intestinal toxemia," will cause such symptoms as headache, fatigue, anorexia, depression and vague pain. These may well be largely due to the associated conditions of neurasthenia and physical inferiority.

On palpation of the abdomen, gurgling of gas is felt, and frequently a full rounded cecum filled with feces, which may be somewhat tender, is found. Occasionally the whole colon is palpable. X-ray examinations show an atonic dilated colon, which empties slowly and often contains an excess of gas, especially in the flexures and cecum. The x-ray shows that the atony is frequently associated with ptosis which especially involves the cecum. The large cecum, heavy with contents, slides deep into the pelvis in the erect position, and the flexures of the colon are close to the crest of the ileum. Atony of the cecum may be combined with spasm of the transverse colon.

Cases of acute paresis of the bowel, or paralytic ileus, are an entirely different group, surgical in type and can be only briefly considered here. They occur chiefly after laparotomy, shock, trauma, or peritonitis. The paralysis after operation is a result of handling, drying or cooling the bowel, or of infection. It is a common nuisance after laparotomy, and is the source of the "gas pains" due to distention of the atonic bowel with fermenting contents, and may be very serious or even fatal. In the more serious cases the symptoms resemble those of intestinal obstruction, gaseous distention of the bowel with abdominal tympany, rigidity, pain and tenderness, retention of gas and feces, vomiting, weak pulse and general collapse.

Diagnosis.—The diagnosis of atonic constipation is not difficult. The history of ptosis, fatigue, old age, toxemia or neurasthenia, the presence of an overfilled cecum or colon together with x-ray findings above described are conclusive. However, it is important that atony and paresis of the bowel, both acute and chronic and especially the former, be not confused with organic obstruction by adhesions, twists, tumor, etc. In the paralytic cases there is a history of lapartomy, peritonitis, or shock, no visible peristalsis of the bowel, and no organic cause of obstruction.

Treatment.—The chronic cases need a general **tonic treatment, rest, graduated exercises, gymnastics** and **outdoor sports.** This does not affect the bowel directly, but the intestinal muscles and nervous control take part in the general improvement. **Abdominal massage** and **high frequency currents** are used to stimulate and tone up the bowel. Associated conditions such as ptosis should be treated by **abdominal exercises** or by a **supporting belt** or **corset.**

The treatment of constipation combined with atony will be given in another chapter. In general it may be said that it is not wise to overload the limp bowel with coarse vegetable residue, which is likely to ferment and cause trouble; the constipation may be relieved, but the patient pays too great a price in flatulence and distress. The diet should contain only a moderate amount of vegetable and fruit pulp to which may be added drinking water, buttermilk, mineral oil, agar, and rectal injections. We always wish to wean the patient gradually from chronic drug habits, but sometimes a small drug stimulant such as **cascara, rhubarb,** or **senna** may be better than a large mechanical stimulus of indigestible cellulose. This is especially true of delicate and elderly persons.

Paralytic ileus after operation is frequently very serious and requires special treatment by **rectal tube, rectal injection, turpentine stupes,** or even by **surgical drainage** of the peritoneum or bowel. Pituitrin or eserin given subcutaneously have little effect.

ENTERALGIA, HYPERESTHESIA, PARESTHESIA OF POWEL

Enteralgia is a true primary neuralgia of the intestinal nerves, with all the characteristics of neuralgic pain. It may involve the whole mesenteric plexus or part of it, just as a sciatica may involve the whole or part of the sciatic nerve. The other abnormal sensations consist of pressure, fullness, burning, etc., or the sensation of a foreign body in the bowel.

Etiology.—Disordered sensations or pain in the bowels are symptoms of many diseases. There is a small group of purely nervous origin found in neuropathic persons, analogous to the chronic neuralgia seen in other nerve tracts. The more carefully such a group of cases is studied, the fewer will be called enteralgia. It should be distinguished from the pain due to intestinal inflammation and from the colic due to increased tension of the gut or to violent contraction of the intestinal muscles. One of the most important causes of enteralgia is lead poisoning; another is tabes with abdominal or anal crises. It is possible that gout may cause enteralgia.

Symptomatology.—The patient with enteralgia complains of attacks of cutting, boring, or burning pain in the abdomen, especially in the lower half. The pain may appear and disappear suddenly and be very severe. Tabetic crises are typical examples of these painful attacks which may last from a few minutes to many hours. There is a striking contrast between the severity of the pain and the negative findings on physical examination of the abdomen. Between attacks the digestion appears normal.

Normal persons have no unpleasant sensations as a result of digestion.

Neuropathic cases may have peculiar or disagreeable feelings of burning, fullness, or the "sensation of an intestinal parasite," or as if a foreign body has been swallowed. In another type of cases, there is lack of sensation in the rectum, a lack of the normal "call to stool," which is probably sympatheticotonic and results in chronic rectal constipation.

The physical findings are those of vague or occasionally localized abdominal tenderness, with the stigmata of neurasthenia, or hysteria, nervousness, hyperesthesia, and active reflexes, or signs of lead poisoning, tabes, and the like.

Diagnosis.—The diagnosis of nervous enteralgia may be easily made when tabes, lead poisoning, neurasthenia or hysteria is present. In the absence of these conditions the diagnosis of nervous enteralgia is always doubtful, and the pain is usually due to some organic cause, such as disease of the liver, gall-bladder, genito-urinary tract, or organic diseases of the bowel, such as colitis, hernia, obstruction, appendicitis, etc.

The commonest obscure causes of intestinal pain are mild and undercovered intestinal catarrhs, diseases of the spinal cord, occasionally arteriosclerosis (abdominal angina). Many cases, no doubt, have an organic cause which is never found, such as stretching the mesentery, brief local inflammation, parasites, etc.

Treatment.—The general treatment is that of the underlying cause, such as lead, tabes, neurasthenia, etc. The immediate treatment is that of neuralgia, namely, **local hot applications** and 10 grains (.65 gram) of **acetphenetidin, acetylsalicylic acid** or **triple bromids**: **opium,** on account of habit formation, should be used only for severe pain. Some cases suggesting vascular spasm yield at once to **nitroglycerin,** 1/50 to 1/100 grain (.0013 to .00065 gram). For the **painless abnormal sensations sedatives** and **psychotherapy** are used.

INTESTINAL FERMENTATIVE DYSPEPSIA

This rather uncommon disease is characterized by poor digestion of starches in the bowel, while proteids and fat are well cared for; this is associated with mild abdominal discomforts and an almost normal physical examination. The pasty, acid stools are quite characteristic.

Etiology.—This condition was first described by Schmidt and Strassburger who consider it to be a functional disturbance of intestinal secretion, a lack of the *succus entericus,* or inability to digest cellulose, with some increased emptying of the bowel, resulting from acidity of the bowel content.

Symptomatology.—The patient complains of pasty or foamy, light yellow, irritating, sour stools, often three or four a day, with passage of much sour gas per rectum. Sometimes the gross feces appears normal, but the incubation test always shows pronounced carbohydrate fermentation. Microscopic examination of the feces shows an increased amount of starch (poor starch digestion). There is general abdominal discomfort with gaseous distention and often some pain, diminished appetite, and general malaise. The general condition is usually quite good. Gastric secretion and digestion are entirely normal. There are often general nervous symptoms, such as poor sleep, headache, and fatigue.

The abdomen is full, sometimes tense, with gurgling of gas on palpation and some diffuse tenderness. The examination of the stools is most important. They are bulky, pasty or liquid, light colored, acid, frothy without mucus, and contain undigested starchy food. Microscopic examination, best done after use of a Schmidt-Strassburger test-diet for a few days, shows many undigested starch cells, staining deep blue with Lugol's solution. If the fresh feces are incubated for twelve hours, great quantities of gas develop and the stools become very acid.

Diagnosis.—The diagnosis is easily made from the character of the stools,—foamy, acid, soft, light colored, with great excess of undigested starch, associated with the patient's complaining of intestinal discomforts and flatulence, without gastric symptoms or changes in gastric secretion.

Treatment.—The treatment is satisfactory as a rule, and is chiefly **dietetic.** The diet resembles the old diabetic diets: starches are reduced to a minimum and proteids and fats used freely. The patient begins with milk, broths, eggs, gelatin, butter, fish, tender meats, cheese, custard, ice cream, and adds gradually, after a few days, cooked fruits, toast, zwieback, well-cooked cereals and starchy puddings. Potatoes are omitted for a long time, as well as the coarser vegetables, such as beans, corn, cucumbers, beets, parsnips, carrots, turnips, cabbages, onions, radishes, etc. Small feedings six times a day are the rule. Diet and good hygiene are far more important than drugs. **Bismuth salicylate** in 10 grain (.065 gram) doses three or four times a day is sometimes useful; also **bismuth subcarbonate,** 1 teaspoonful, with **tannigen,** 10 grains (.065 gram), three to six times a day, if there is active diarrhea. **Taka-**

diastase in 15 grain (.972 gram) capsules after meals has been used to supplement the lack of the starch-digesting ferments.

Prognosis.—The prognosis is usually good if the patient will diet strictly for a long time, omitting coarse vegetables (cellulose), and restricting the starches. The power of digesting starches and cellulose gradually returns as a rule, and the diet can be increased by easy stages without recurrence. In some cases there is a persistent limit to carbohydrate tolerance as in diabetes which must not be exceeded.

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CHAPTER XXXIV

ACUTE AND CHRONIC PERITONITIS AND TUMORS OF THE PERITONEUM

By Alexius McGlannan, A.M., M.D.

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ACUTE PERITONITIS

Anatomy.—The peritoneum is a serous sac which lines the abdominal cavity and surrounds in whole or in part the viscera. Its surface consists of delicate mesothelial cells resting on a tunica propria of fibroelastic tissue. A layer of subserous, areolar tissue connects this membrane with the adjacent structures. This tissue varies greatly in density and quantity in different regions. For practical purposes the transversalis fascia and the rest of the subperitoneal tissues are best considered as one structure with the peritoneum itself.

Parietal and visceral layers are distinguished, and the great omentum is a reduplicated layer of serous membrane which hangs from the stomach and colon free in the peritoneal cavity. Nerves, blood-vessels and lymphatics run in the subserous areolar tissue and make anastomoses with corresponding structures in the viscera, as well as with those in the walls of the abdominal cavity. In general the two sets of communications are distinct, the visceral being through the various mesenteries, while those of the parietal layer take place by direct contact with structures in adjoining tissues. The intraperitoneal lymphatics communicate directly with those of the pleura through the diaphragm and empty into the anterior mediastinal and lumbar lymph glands.

In the male the peritoneum is a closed cavity, while in the female there is an external communication through the Fallopian tubes. The infoldings and reflections of the peritoneum give it a surface area about equal to that of the skin.

The nerve supply of the peritoneum and that of the contained viscera is intimately associated with the nerve supply of the abdominal wall. The six lower intercostal nerves which supply the anterior abdominal wall, are also distributed to the diaphragm. The same nerves with the ileo-inguinal and ileo-hypogastric supply the peritoneum either directly or through the sympathetic plexuses.

The parietal peritoneum is extremely sensitive, while the visceral layer has no ordinary sensitiveness. Rough handling of the visceral layer causes pain by making traction on the parietal layer. The localized pain and muscle spasm characteristic of inflammation of intraperitoneal viscera is explained by the peculiar relation of the nerve supply of the viscera and that of the abdominal wall. When the inflammatory lesion has become extensive involvement of the parietal peritoneum by lymphagitis, or other modes of extension, gives another type of tenderness.

Definition.—Peritonitis is an inflammation of the peritoneum, which may be acute or chronic.

Pathogenesis.—Practically all cases of peritonitis are the result of the introduction of bacteria into the cavity. The invading organisms may enter by way of the blood or the lymphatics, but as a rule their entrance is either through a wound of the abdominal wall, or a perforation or rupture of one of the contained viscera. This perforation may be the direct result of traumatism, but more often it occurs in the course of an acute infectous disease of the organ involved. The infection may take place by extension from a focus in the subperitoneal tissue, but the reaction in this tissue is usually so vigorous that the infection spreads along the fascia behind the peritoneum to a point on the skin surface. The open Fallopian tube is a frequent portal of entry in cases occurring in women.

The effect of an invasion varies. The healthy endothelial cells may destroy many bacteria and no evidence of the conflict remain. There may be in addition exudation of fluid and migration of many phagocytotic leukocytes and yet the process resolve without structural change in the peritoneum and without marked constitutional disturbance. On the other hand, the exudate may remain unabsorbed or increase in quantity, and the process pass into one of chronic peritonitis.

When injured the endothelial cells lose their luster and the surface of the peritoneum becomes dull and roughened, dark red in color, edematous, and flaked with deposits of fibrin. Intense congestion with small petechial hemorrhages takes place in the subserous layer, and actual bleeding into the cavity may occur from an eroded vessel. Migration of leukocytes and exudate of serum occur. The serum is bacteriolytic and may be fibrinous or seropurulent. A seropurulent exudate is more common in peritonitis than is the case with infections of other serous membranes. The plaques of fibrin may become necrotic and sloughing off leave exposed a surface of unprotected blood and lymph capillaries through which rapid absorption of toxins and bacteria may lead to a fatal outcome.

Similarly the destruction of endothelium forms raw surfaces through which absorption occurs, unless two adjacent raw areas adhere. As the endothelial cells suffer from injury the retroperitoneal tissues become thickened and indurated by the migration of leukocytes and the other products of inflammatory reaction, in this way making an attempt to protect the body as a whole from the products of the infection.

Physiology.—Normally there is a secretion of fluid, principally by the great omentum, which moistens the surface of the peritoneum and forms a small quantity of fluid free in the cavity. The peritoneum possesses great power of absorption. Liquids are absorbed directly into the blood stream, while solid particles are absorbed into the lymphatics after they have been taken up by the phagocytes. Colloid solutions are absorbed less rapidly than those of crystalloids, and the absorption through the lymphatics is slower than that through the blood stream. Both parietal and visceral layers, as well as the omentum, are active in the process of absorption.

The absorption of solids is delayed by the resistance of the serosa cells, and if circumstances permit, much of the foreign material is encapsulated rather than absorbed. When the lining cells have been destroyed by traumatism, absorption of solid material becomes more active. An intact layer of lining cells, therefore, becomes an important agent in protecting the organism against the effects of bacteria which may invade the peritoneal cavity.

The endothelial cells surround the bacteria by phagocytotic action, carrying them to the lymph glands, and thereby protect the circulation from direct invasion. Wherever the endothelial cells have been destroyed by traumatism or other agents, the bacteria are able to enter into the blood stream directly and in this way produce asepticemia.

Absorption from the peritoneum has been the subject of much experimental study. At the present time the results may be summarized as follows:

(1) Absorption in the region of the diaphragm takes place through certain specially constructed areas of the peritoneum and the lymphatics which run on the under surface of this muscle.

(2) An intact omentum adds to the rapidity of absorption.

(3) Solutions of certain substances (*e. g.*, sodium chlorid, dextrose), if introduced in an aseptic manner, will be absorbed without any harmful action on the peritoneum.

(4) Absorption is more rapid from the upper (diaphragmatic) than from the lower (pelvic) area.

(5) The position of fluids in the peritoneum is influenced by gravity and by the presence or absence of adhesions.

Localized Peritonitis.—In localized or circumscribed peritonitis the area of primary involvement is limited and the development of a fibrinous exudate leads to the agglutination of adjacent structures which form a barrier to the extension of the infection. This fibrinous exudate is rapidly organized to form fibrous adhesions. Under favorable circumstances the bacteria contained in such a walled-off area become attenuated, the pus may become sterile, and small abscesses even may be absorbed.

After such a localized peritonitis has healed either spontaneously or after appropriate surgical treatment, the adhesions become thinner and thinner and many times finally disappear.

Diffuse or Spreading Peritonitis.—With primary involvement of a wide area of peritoneum, especially if this be associated with marked traumatism of the endothelium and the presence of foreign material, there will be extensive hyperemia of the surface with a large seropurulent exudate and great absorption of bacteria and toxins into the circulation. The result of such a process depends entirely upon the relative resistance of the patient and the virulence of the infection. Death may occur in from twenty-four to thirty-six hours, or the spreading infection may continue for days before the fatal termination. The pumping action of the diaphragm and the peristaltic movements of the intestine are factors in spreading the infection.

Agglutination of intestinal coils to one another and to the omentum and parietal peritoneum are the most potent factors in combatting its extension. As the infection injures the endothelium of the intestine distention of the gut occurs, either from toxic damage to the nerve supply or direct action on the muscle. This paralytic ileus, although usually considered a bad feature of the disease, may be part of the defense. Peristalsis is arrested in the distended loops of bowel, and while the absorption of accumulated material from such loops is considered dangerous, it is certain that it does not compare in seriousness with the toxemia resulting from a mechanical obstruction associated with vigorous peristalsis.

As the peritonitis subsides in the cases which end in recovery the exudate will form single or multiple collections surrounded by adhesions. These smaller exudates may become sterile and be absorbed or they may persist as single or multiple abscesses which will require surgical treatment. The adhesions usually disappear soon after the exudate. Recovery from acute peritonitis when it occurs is usually complete in the sense that the endothelial lining is regenerated and the functions of the peritoneum restored. Should the abdomen be opened a year or more after a patient has recovered from a diffuse peritonitis, as for example, to repair an incisional hernia after operation for perforative peritonitis, one is struck by the smooth glistening normal appearance of the membrane, the absence of adhesions, the thorough inclusion of ligatures, sutures, etc.

Etiological Factors.—As has been mentioned most cases of peritonitis are secondary to perforation, or rupture of an abdominal viscus. The greatest single factor in the production of peritonitis undoubtedly is leakage from a diseased appendix. Acute appendicitis seldom offers any great difficulty in diagnosis and facilities for prompt and skillful removal of the organ are nearly always accessible, therefore prophylactic surgery could reduce the frequency of peritonitis to a great degree.

Other common causes are perforation of a gastric or duodenal ulcer, leakage from a diseased gall-bladder, perforation of a typhoid or tubercular ulcer of the intestine and sloughing of a malignant tumor. In women salpingitis and direct infection through the open tubes frequently set up the condition. The route of invasion in a case of puerperal peritonitis may be direct through the tubes, or by diffusion through the lymphatics, or be secondary to some previously localized focus.

Penetrating or perforating wounds of the abdomen and subcutaneous rupture of a viscus will cause peritonitis of varying degrees according to the circumstances surrounding the injury. Damage to the blood supply of the intestine by trauma, either open or subcutaneous, or by vascular disease leads to an escape of septic material through the devitalized wall of the bowel. It is important to remember that the effect of subcutaneous trauma on the blood vessels may not become manifest until twenty-four or seventy-two hours after the injury.

Great distention of an obstructed or paralyzed bowel will cause similar leakage. The rupture of aseptic organs or growths and intra-abdominal hemorrhage set up a form of peritonitis milder than that caused by bacterial infection. Hematogenous and lymphatic invasion occur, but the frequency of infection by these routes is much less than that of the perforative types. Pneumonia, influenza, and septic sore throat are the usual primary diseases. Cases of hematogenous peritonitis have been described following erysipelas and scarlet fever.

The bacteria involved in peritonitis are numerous, and in most cases the infection is a mixed one. In point of frequency of occurrence the colon bacillus stands first. Other common forms are the streptococcus, pneumococcus, bacillus pyocyaneous, bacillus typhosus, bacillus tuberculosis, gonococcus and staphylococcus. Among the rarer forms of bacteria found in peritonitis are the proteus and the gas bacillus.

The colon bacillus seems more prominent in the perforative types of peritonitis, while the streptococcus or staphylococcus is usually the predominant organism in the cases produced by a wound which opens the abdomen. Puerperal peritonitis is practically always due to a streptococcus infection. Pyocyaneous infection occurs in perforative peritonitis. This bacillus appears to have an antagonistic action on the colon group, because the two types are never associated in a case of peritonitis. The blue pus is not seen within the abdomen but only on the surface where contact with the air is possible. The local and systemic reactions to the pneumococcus, gonococcus and the tubercle bacillus are fairly distinct and will be considered in detail later.

Symptoms of Acute Peritonitis.—The cardinal symptoms of peritonitis are abdominal pain with nausea or vomiting, local tenderness, spasm and rigidity of the abdominal muscles and change in the temperature and pulse rate. Varying degrees of shock may accompany the onset of the disease.

Pain.—Pain is the most constant of the symptoms of peritonitis; it is also the most constant symptom of those lesions which cause peritonitis. Persistent abdominal pain, therefore, should never be dismissed as an unimportant condition.

The pain of onset may be relatively slight and gradually increase to great intensity as the process of inflammation advances, after which it may remain stationary, or decrease. In certain rare cases the pain may never be severe enough to give the patient serious concern, and this in spite of the fact that the peritonitis is spreading and the constitutional effects of the septicemia are of the gravest type. In the perforative types intense pain is the characteristic symptom of onset.

The pain is continuous, with or without exacerbations. It is not often paroxysmal, although occasionally the etiological factor in the development of the peritonitis may also be responsible for a simultaneous mechanical obstruction of the bowel, in which event the pain would become paroxysmal. Such a condition may be noted in cases of appendicial peritonitis in which the edematous ileum has been acutely angulated by the developing protective adhesions. Similarly, paroxysmal pain may result from spastic contracture of the bowel, occurring as a reflex from the irritation of the peritoneum by the infecting material. Exacerbations probably represent the alternate contraction and relaxation of an inflamed organ and the intermittent flow of irritating material into the peritoneum.

The severity of pain bears no relation to virulence of infection. A patient with an overwhelming streptococcus peritonitis may have little or no pain. Spontaneous relief of pain may accompany the development of gangrene, or it may take place after a tense localized abscess has ruptured. As a rule relief or subsidence of the initial pain followed by its sudden return after an interval indicates rupture of the primary focus into the general peritoneal cavity.

Shock.—Shock of a greater or lesser degree accompanies the pain of perforation. Shock is usually marked in the traumatic cases and is especially severe in perforations or ruptures of all kinds when they are accompanied by hemorrhage.

Nausea and Vomiting.—The initial vomiting is a reflex phenomenon. The combination of nausea or vomiting with abdominal pain, sometimes spoken of as peritonism, is observed in practically all cases of peritoneal irritation irrespective of the cause. In the presence of these symptoms the greatest alertness is demanded until the attending physician has determined the cause for their development.

The vomiting takes place without regard to the ingestion of food, although it is generally increased by any material taken into the stomach. At first it consists of ingested food or gastric juice, later it may become bile-stained. As the peritonitis develops the character of the vomitus is likely to change with the severity and extent of the infection. The material becomes green, brown and black from regurgitation of decomposed material from the intestine. In certain severe infections hemorrhage from the gastric mucosa stains the contents with partially digested blood.

When the peritonitis becomes localized, vomiting usually stops. Recurrence of vomiting, like recurrent pain, means a new extension of infection. The initial vomiting is forceful, but late in the disease there is regurgitation from the dilated stomach rather than true vomiting. The dark material expelled has a disagreeable taste and odor and is extremely irritating to the mouth and skin of the face.

HICCOUGH.—Hiccough may occur in place of the initial vomiting and has the same significance. Later in the disease hiccough may be caused by pressure of the distended bowels or by infection of the diaphragm. This late hiccough is a bad sign.

TENDERNESS.—Pain on pressure is a general sign of inflammatory reaction. Unless the connection with the central nervous system has been broken, pressure on an inflamed area produces pain and with it some kind of defensive muscular reaction. In peritonitis the tenderness varies from an exquisite degree which makes contact with the bed clothes painful to a mild discomfort when pressure is made over the involved area. As a rule however, there is prompt response to pressure over the inflamed area evidenced not only by voluntary complaints of the patient, but also by the reflex contraction of the abdominal muscles protecting the diseased area. A certain degree of tenderness is found all over the abdomen, but in the early stages the tenderness is always most marked in the region where the peritonitis begins. Apparently tenderness is largely the result of friction on an inflamed surface and therefore it becomes less acute as an exudate forms and protects such an area. With localization of peritonitis, tenderness becomes limited to the region involved.

Localized pain on distant pressure sometimes indicates the seat of a circumscribed peritonitis. This sign is credited to Rovsing who explains its occurrence as the result of pressure on the inflamed surface transmitted from the hand of the examiner by way of the column of gas contained in the bowel.

In some cases there is no tenderness when firm gentle pressure is made over the diseased area, but pain is felt when the pressure is suddenly removed. Both of the last-mentioned methods of observation are more valuable in determining the presence of a local, relatively quiet focus than in making the diagnosis of an active acute process.

MUSCLE SPASM AND RIGIDITY.—These conditions of the abdominal wall represent a protective mechanism to restrict the movements of the inflamed peritoneum. Limitation of respiratory movements of the abdomen, or confining respiratory action to the costal type, is a further evidence of this protective muscular rigidity. Contracture of the ileopsoas muscle leads to flexion of the thigh.

Muscle spasm and rigidity, being phenomena of reaction, depend in a degree upon the character and extent of the infection and the resistance of the patient. At the onset the spastic contracture is usually general and equal all over the abdomen. As the inflammatory change develops the rigidity becomes most marked in the segment of the abdominal wall covering the primary focus. With localization of the peritonitis there is also localization of resistance with relaxation of the muscles over the uninvolved peritoneum.

When the patient is strong and vigorous rigidity is prominent and persistent, the muscles may contract so firmly that the abdomen is board-like and retracted. This sign is especially common in the early stages of peritonitis following rupture of a peptic ulcer. When the abdominal walls are thin and flaccid, as in the cases of multiparous women, spasm of the stretched out muscles may produce so slight a degree of rigidity that it is hardly appreciable. In certain types of peritonitis, especially non-perforative varieties in which there is a slowly developing fibro-purulent exudate, muscle spasm and rigidity may never be pronounced. Fulminating infections, and those associated with gangrene of the viscera, are likely to be associated with such severe toxemia that the muscular reaction is feeble or even may be abolished.

Muscle spasm, therefore, is a valuable sign when it is present, but absence of rigidity must not be considered an indication that peritonitis also is absent. When present the spastic contracture of muscle almost invariably persists even when the patient is anesthetized.

Changes in Temperature and Pulse Rate.—Variations in the temperature are usually an index of the patient's resistance to the infection. As a rule there is an elevation which may seem slight when compared with the intensity of the other symptoms. Such moderate fever must not be misinterpreted in diagnosis and prognosis. Fever is a fairly accurate measure of resistance against the toxins absorbed. Low or subnormal temperature is the rule at the onset of severe cases. High fever is common when there is marked invasion of the lymphatics as well as of the endothelial surface.

The temperature should always be taken with the thermometer in the rectum. The cold skin and the open mouth of peritonitis may register low or subnormal temperatures at the same time that the rectum shows a rise as high as 104° F. (40° C.).

Occasionally, especially in cases due to rupture of a gangrenous organ, a chill may take place at the onset of the attack. The chill is seldom repeated, except in cases of puerperal peritonitis. Recurrent chills indicate a pyemic process with the development of new areas of infection.

Change in the pulse rate is a reliable sign in peritonitis. From the onset, in most cases, the pulse rate is increased and its volume diminished. Occasionally at the onset the pulse becomes fuller, but this condition never lasts long and the pulse soon becomes the characteristic peritonitis type, that is, small, wiry and rapid.

The increase in pulse rate may develop so speedily that a progressive rise will be noted in observations repeated at 10-minute intervals. At the onset the change in rate is reflex through irritation of the vagus nerves; later, myocardial changes are produced by the action of the toxins on the heart muscle.

Blood-pressure.—Shock incident to the rupture of a viscus or a walled-off abscess will cause a fall in blood pressure. If the infection is a fulminating one, the low pressure will continue; but, if the patient rallies the blood pressure rises and reaches a point above the normal, where it remains during the height of the peritonitis. In 23 cases Crile found an average pressure of 162 at the height of the disease. The rise in blood-pressure was distinct in the cases of peritonitis due to typhoid perforation. The blood-pressure is raised at the time of the attack in tabetic crises and in lead colic. In all other forms of sudden severe abdominal pain it is lowered.

Cyanosis may result from direct embarrassment of the heart, but usually indicates a severe toxemia. The bluish tint shows first in the finger nails. Jaundice may be extensive or limited to a slight yellowing of the sclerotics. It is a sign of severe infection.

DISTENTION.—Distention of the abdomen is due to the accumulation of gases in the bowel (meteorism), or to the presence of gases free in the peritoneal cavity (pneumoperitoneum). The flatulent distention of the bowels is a relatively late symptom resulting from changes in the intestinal wall which inhibit peristalsis. Pneumoperitoneum, on the other hand, may be among the earliest symptoms because of the escape of gas through perforation of the bowel. This is particularly likely to occur in cases of traumatic rupture of the intestine.

Another variety of pneumoperitoneum is that found in cases of peritonitis due to infection with one of the gas-producing organisms. As a rule the presence of gas free in the cavity is recognized by the general tympany with obliteration of the normal area of liver dullness. When meteorism is extensive the pressure of the distended bowels may push up and rotate the liver so that the area of dulness in front may be lost; but the dulness in the mid-axillary line is very slightly diminished, as long as the gas remains in the bowel. When the liver dulness in the mid-axillary line disappears, the diagnosis of gas free in the peritoneal cavity should be made.

FLUID.—Fluid in the peritoneal cavity may be that of the inflammatory exudate alone, or may consist of blood and other material extravasated from a ruptured viscus, which in turn is mixed with more or less of the inflammatory exudate following the rupture.

Shifting dullness in the flanks indicates fluid free in the cavity. Fixed dullness points to an encysted collection of fluid. Ransohoff called attention to a saffron-yellow discoloration of the umbillicus which was present in a case of peritonitis where the exudate was deeply stained with bile. Cullen found the umbillicus stained bluish-black in a case of ruptured extra-uterine pregnancy with a great quantity of blood in the peritoneal exudate.*

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*Denzer describes a method for the diagnosis of peritonitis by means of abdominal puncture. A specially constructed trocar and canula fitted with capillary glass tubing is made for this purpose by the Randall Faichney Company of Boston, Mass. The diagnostic puncture is made over the area of exudate, care being taken to avoid a distended bladder or one of the solid viscera. The trocar is withdrawn and the capillary tube inserted as far as it will go. Fluid rises in the glass tube at once or after a few minutes. The gross appearance of the fluid, as well as its examination by the microscope and by cultural methods gives information of diagnostic value. The method has been used only in the peritonitis of infants.

In rare cases the infection may extend to the structures of the abdominal wall. In such an event a local edema develops which may be sufficiently extensive to lead to pitting. A purulent peritonitis may find a spontaneous opening at the umbillicus. The condition is rare. Of 13 cases noted by Cullen 12 were in young children, 11 of them being girls.

LABORATORY EXAMINATIONS.—*The Urine.*—The effect of the toxemia on the kidneys is shown by the presence of albumin and varying quantities of casts in the urine. The occurrence of similar changes in the urine in so many other conditions makes the finding of albumin and casts of no diagnostic value. The same is true of indican, etc., whose presence in the urine simply indicates abnormal destruction of protein and is not at all limited to cases of peritonitis.

Bacteria of the same type as those present in the peritoneal exudate have been grown from the urine. This fact is interesting, but as yet it has not proved of practical value in the diagnosis or treatment of the disease.

The Blood.—(1) *Leukocytosis.*—Increase in the number of white blood corpuscles is common in peritonitis. The average leukocyte count is from 15,000 to 18,000, varying from 10,000 to 30,000. The higher counts are more common in hematogenous infections than in perforative peritonitis. The greatest leukocytosis seems to occur in the pneumococcus cases. Well-marked suppurative peritonitis may exist without any accompanying leukocytosis. In tuberculosis peritonitis there may be a leukopenia, and the number may be below normal in peritonitis from perforation of a typhoid ulcer. In the typhoid cases however, if the patient is able to make any reaction to the infection of the peritoneum, the hourly count will show a rising leukocytosis, and of the relative proportion of polyneuclear neutrophil corpuscles. The increased proportion of polyneuclear cells, as shown by the differential leukocyte count has greater significance than the total increase of white cells. Interpreted with consideration of other symptoms, fluctuations in the leukocyte count, may be of some prognostic value. Like the temperature curve, that of the leukocytes generally runs parallel with the extent of infection and the reaction of the patient. As the absorption of toxins is diminished by limitation of the peritonitis, there is usually a drop in the leukocytosis with distinct improvement in the other symptoms, notably in the pulse rate. On the other hand it must be kept in mind that exhaustion of the patient's resistence may be indicated by a falling leukocytosis, and that a diminution in the number of white cells in the blood can never be considered proof that infectiousness of the exudate is at an end.

(2) *Anemia.*—A fall in the number of red blood cells is usually noted in peritonitis, which decrease may become marked in severe cases.

(3) *Blood Cultures.*—In spite of the very clear pathological evidence that bacteria are absorbed directly into the circulation in peritonitis, blood cultures taken during life practically always remain sterile.

(4) *The Non-Protein Nitrogen in the Blood.*—An increase in the

non-protein nitrogen in the blood is found to take place in peritonitis as well as in several infections. The quantity is never so great and the accumulation progresses at a much lower rate than that noted in cases of acute intestinal obstruction.

The significance of age, sex, and previous history is more important in distinguishing between the different sources of peritonitis than in recognizing the disease itself. The hematogenous varieties occur most often in children and in old people. Appendicial peritonitis is common to all ages and both sexes, and is the most frequent variety in any large collection of cases. A history of previous dyspepsia will suggest peptic ulcer as the causative factor, especially in young adults of either sex. In older men perforation of a cancer may be the cause. Pelvic inflammatory disease is responsible for many cases in women. After the age of forty the probability of the gall bladder being responsible for the peritonitis must be considered.

Clinical Course.—The patient with peritonitis always is manifestly sick. The onset of the perforative cases with severe pain and shock is alarming and a speedy call is made for relief. The hematogenous cases are associated with signs of severe general infection. Soon after the onset some disturbance of the gastro-intestinal tract occurs. This is usually nausea or vomiting, but may include diarrhea or constipation. The pain directs attention to the abdomen and the presence of muscle spasm and rigidity with tenderness is seldom difficult to recognize. The temperature may be subnormal during the period of shock immediately following rupture of a viscus, but fever of some degree develops as the patient rallies from the initial depression. The rectal temperature is the only reliable measure of fever in peritonitis. The pulse becomes rapid, and wiry, soon reaching a rate of 100 or more.

The muscle spasm prevents the full excursion of the respiratory muscles, in consequence of which the breathing becomes shallow and its rate increased. Unless the infection is a fulminating one, movements of the alae nasi with respiration do not occur until late in the course of the peritonitis.

The patient takes an almost characteristic attitude in bed. The knees are drawn up to fix the psoas muscles, while the respiratory movements become entirely or principally thoracic, because of the rigidity of the abdominal wall and limitation of the movements of the diaphragm. As the disease progresses the abdomen becomes distended from meteorism and other causes. The increased abdominal tension further embarrasses the action of the heart, already made weak by the action of the toxemia. As a result oxygenation of the blood becomes imperfect and more or less cyanosis develops. A cold sweat makes the skin moist and clammy to touch.

The vomiting takes the form of regurgitation of foul, irritating, dark colored material. Hiccough is often an annoying and uncomfortable symptom of this stage of the disease and is a sign of bad omen.

The urine becomes scanty and high colored and there may be difficulty in emptying the bladder as a result of the action of the infectious material on the bladder wall.

In fatal cases with the spread of the peritonitis distention increases, the pulse becomes faster and more feeble and the respiration shallower, the face grows pinched and drawn, the eyes sunken, the lips blue, the mouth is held open, showing the teeth, usually coated with sordes, and the dry, coated, tremulous tongue. The cyanosis deepens and the extremities become cold. Through it all the mind remains alert, active and rather terror-stricken, although the patient sometimes is hopeful or indifferent instead of anxious. Usually he is restless, tossing his limbs about, but holding his trunk rigid and immobile. Delirium or stupor generally comes on in the last hour or two, the heart's action becomes progressively weaker and death comes with exhaustion of the vital functions.

The rapid deterioration of the entire organism seen in these fatal cases can be compared only with the course of such intense poisonings as snake-bite and cholera. Death may take place within twenty-four hours of the onset of peritonitis, or the disease may run for six or seven days or longer before the fatal termination. In most cases the third day seems the critical time, and unless improvement begins to be evident at the end of seventy-two hours, there is great probability that the patient will die.

Localization or recession of the infection mitigates the symptoms. Changes in the character and rate of the pulse are the best signs of decreasing toxemia. A spontaneous movement from the distended bowel brings great relief and is a most favorable sign. The vomiting becomes less frequent, or ceases entirely; the general muscle spasm relaxes, leaving only a segment of the abdomen rigid. Pain and tenderness are less marked and are limited to the area of involvement; the temperature gradually falls; the dry tongue begins to moisten; the skin takes on a better color and feels warm. A profuse hot sweat often initiates the improvement in the character of the symptoms.

Differential Diagnosis.—The combination of symptoms, pain and gastric disturbance with more or less shock is the common onset of a great variety of intra- and extra-abdominal diseases.

Most of the intra-abdominal lesions are potential sources of peritonitis and therefore demand early recognition and practically always prompt surgical operation, in order that the pathological process may be corrected or the diseased area excised before the peritoneum becomes seriously involved. It is of the greatest practical importance, therefore, to separate the conditions which urgently require operation from those in which operation may be delayed, or in which it is not necessary.

ABDOMINAL SYMPTOMS OF THORACIC DISEASE.—The onset of pneumonia, of diaphragmatic pleurisy, of pulmonary infarction, of pericarditis, as well as the acute exacerbations of cardiovascular disease, may all be ushered in by marked abdominal symptoms. Throat infections in children, especially those associated with exanthematous diseases may mimic peritonitis in their symptoms of onset.

PNEUMONIA AND PLEURISY.—Abdominal symptoms may come on at any time in the course of the disease, but confusion is most likely to take place when they occur at the onset of the thoracic disease. The greater relative frequency of respiration as compared with pulse rate indicates the thoracic disease rather than peritonitis.

Superficial tenderness is more marked than the deep tenderness in the thoracic lesions and if this area of pain and tenderness be mapped out carefully and the patient then be directed to hold his breath, the pain and tenderness, as well as the muscle spasm, will be absent as long as the diaphragm remains quiet.

In the absence of other signs of acute thoracic disease abdominal pain associated with movements of the alae nasi during respiration indicates a thoracic and not an abdominal lesion. Respiratory movements of the nostrils come on late in peritonitis. The anxiety in thoracic disease is lethargic and resigned, not terror-stricken and active as in abdominal disease. The average leukocytosis of pneumonia is about 30,000, much higher than the average in peritonitis.

Cardiac Lesions.—Acute peri- and endo-carditis, as well as exacerbations of chronic cardiovascular lesions may be initiated with intense abdominal symptoms. Changes in the size and position of the area of cardiac dullness and in the position and character of the apex beat should focus attention on the heart and pericardium as the source of the symptoms. A small thready pulse, with slight increase in the rate and a regular rythm, but quite distant heart sounds, indicates a cardiac and not an abdominal lesion. The same is true of the development of a slow pulse and fainting attacks with the abdominal pain. If the patient is over forty, with a history of dyspnea, and if there are physical signs of cardiac enlargement, the probability of a thoracic rather than an abdominal cause for the symptoms becomes greater.

When the apparatus is available, x-ray examination of the chest will often show the lung lesion and will aid in recognizing the cardiovascular ones when physical signs are indistinct. The same is true of the electrocardiogram, which may add to the certainty of the heart as the cause of the symptoms.

If the possibility of thoracic disease as the cause of the symptoms is kept in mind, a mistake in their interpretation is unusual. During an epidemic of lung infections such mistakes are rare; but at other times a distinction between peritonitis and thoracic disease may be extremely difficult. This is particularly true in the case of children, and few surgeons of experience have escaped the embarrassment of performing an operation in a case of pneumonia, or of postponing one in a case of peritonitis occurring in a child.

Chest Injuries.—The relation of the spinal nerves to those of the peritoneum explains the reflex abdominal symptoms that may follow injury to the chest and back. Referred pain and distention are the commonest. Tenderness is seldom present, although a considerable degree of muscle spasm may exist. Acute dilatation of the stomach, or paralytic ileus may complicate a chest injury giving it a striking resemblance to peritonitis. Usually there is no fever in these cases and the absence of the progressively increasing pulse rate aids in the diagnosis.

THROAT INFECTIONS.—These and the onset of acute exanthemata may be characterized by acute abdominal pain. The pain is almost always localized at the umbilicus, but may be diffuse or located in some other area. Tenderness is never so great as the pain. Delay in the case of the exanthemata brings out the skin lesions and with them a clear diagnosis. In the case of the throat infections delay may bring disaster. Acute gangrenous appendicitis may form a complication of these throat infections, the lymphoid tissue of the appendix being involved with that of the throat in a systemic infection. Acute inflammation of a mesenteric or post-peritoneal lymph gland may be the cause of the abdominal pain in these throat diseases, and suppuration and rupture of such a gland become the starting point of an acute peritonitis.

CRISES OF TABES.—These are often associated with distention, muscle spasm and other symptoms of peritonitis. The characteristic changes in the pupil and in the other reflexes make the recognition of tabes definite. Similar crises resulting from other diseases of the cord are recognized by the signs of the neurological lesion.

VISCERAL CRISES OF ANGIONEUROTIC EDEMA.—These may also resemble peritonitis, although as a rule they are more like acute intestinal obstruction. The history of previous attacks or the association of the symptoms with hives, or purpura, or other superficial manifestations of the disease will make the diagnosis. The occurrence of blood in the urine with the attack is suggestive of the angioneurotic lesion.

LEAD COLIC.—In this disease the abdomen is scaphoid with considerable muscle spasm. The pain is paroxysmal at the onset of the attack, but later the pain becomes constant, although less severe. Tenderness may be present, especially on deep pressure after the paroxysm has ended. In some cases firm pressure gives relief. The pain and other symptoms may be definitely localized on one or the other side of the abdomen. When so localized there is tenderness over the vagus on the same side of the neck; and also contraction of the pupil in the eye on this same side. The pulse is slow in lead poisoning. There is always considerable anemia. The presence of a blue line on the gums is an aid in the diagnosis. The occupation of the patient may give the clue to lead poisoning as the cause of the colic.

ENTEROCOLITIS.—The pain and collapse of acute enterocolitis may confuse this disease with the onset of perforative peritonitis. In the cases of enterocolitis diarrhea is a prominent symptom, and the pain is intermittent. Tenderness is not marked and distention is accompanied by vigorous peristaltic movements. There is a rise of temperature and a rapid pulse, but no progressive increase in the pulse rate.

MUCOUS COLITIS.—This gives symptoms more likely to be confused with those of mild appendicitis than with those of peritonitis. Diffuse pain and collapse may occur. These patients have a history of bronchial asthma or other forms of angioneurosis. There is an eosinophilia shown in the blood count.

OTHER COLICS.—Colic from the passage of calculi, either renal or biliary, is usually quite distinct. Biliary colic is associated with jaundice of some degree and the pain is referred to the epigastrium and toward the right shoulder. In renal colic blood is found in the urine and the pain is referred from the loin along the course of the ureter to the external genitals.

Typhoid Fever.—Pain and tenderness with distention may be present at the onset of typhoid fever, or become marked symptoms of its later course. At the onset these symptoms are seldom so severe as is the case at the onset of peritonitis. Furthermore, the characteristic temperature and pulse curve of typhoid fever is very different from that of peritonitis. Later, in typhoid fever there may be wide spread tenderness and distention but the change in the general condition, and especially in the pulse rate, is not in proportion to the change in the abdominal symptoms. A spreading peritonitis would be accompanied by marked change in the heart's action. In typhoid fever, perforation and subsequent peritonitis is a fairly common complication. The symptoms may be clear enough to make a diagnosis possible at the time of perforation, but the toxemia of the typhoid infection may mask both the perforation and the peritonitis. Pain, tenderness and distention, often with varying degrees of muscle spasm, are common in severe cases of typhoid fever at the stage of the disease when perforation is most likely to occur. Sudden sharp pain, with signs of collapse and the development of rigidity in the lower right quadrant, are most significant signs. A rising pulse rate is an essential symptom, but may also indicate hemorrhage. The value of repeated leukocyte counts has been mentioned. A change from apathy to restlessness associated with the other symptoms is a corroborative sign of great value.

Thrombosis and Embolism of Abdominal Vessels.—Mesenteric occlusion, either arterial or venous, may be embolic or thrombotic. Signs of shock are prominent at the onset of the occlusion. The pain is colicky; tenderness and distention are general; and usually the patient passes a quantity of bloody material by way of the rectum. Preëxisting endocarditis or disease of the vessels is the underlying etiological factor, except in the cases directly due to traumatism.

Thrombosis of the portal vein may complicate ulcerative or suppurative lesions of the bowel or sclerotic disease fo the liver. Severe diffuse pain with signs of shock and distention accompany the sudden obstruction of the vein. In addition there is a bloody diarrhea, jaundice, and tenderness over the course of the vein. When the vessel is the seat of a pyelophlebitis there will be intermittent chills and fever.

Obstruction of the vena cava may complicate any of the acute infectious diseases and give rise to symptoms suggestive of peritonitis. The patients are usually cyanosed to a considerable degree, especially about the flanks and back. The pain is girdle-like. There is considerable distention with moderate tenderness situated in the upper quadrant close to the vertebral column.

Thrombophlebitis may be a primary disease of any of the post-peritoneal veins, or may occur as an extension from a similar process in the veins of the lower extremity. The symptoms are like those described above.

When the process is an infectious one remittent chills and fever are usually marked symptoms.

Abdominal Arteriosclerosis.—This often produces attacks of diffuse angina with distention and nausea. There is absence of peristaltic movement and sound. The region of the diseased vessels is likely to be tender. There is no fever. The presence of sclerotic vessels elsewhere in the body and the relief that comes with return of peristalsis distinguish the lesion.

Aneurysm of the Abdominal Aorta.—This seldom gives difficulty in diagnosis from acute peritonitis, because its symptoms are usually distinct and of a chronic nature. Rupture of an unsuspected aneurysm may simulate the onset of fulminating peritonitis.

Neuroses.—A neurotic individual may develop symptoms very like those of peritonitis. Pain, gastric disturbance and distention may be marked. An elevation of temperature may be shown by the thermometer. Tenderness is always present, but is usually out of proportion to the pressure exerted, and is accompanied by active contracture of the muscles. The muscle spasm and rigidity is confined to the recti muscles. A history of repeated attacks of a similar nature is significant.

Injury of the Abdominal Wall.—Traumatic peritonitis and injuries of the abdominal wall are caused by the same sort of violence and are associated so often that the exclusion of peritonitis after traumatism to the abdomen is a matter of serious concern. The patient is usually shocked and complains of abdominal pain. He may be nauseated or vomit one or more times. The vomitus consists of ordinary stomach contents, or may be a rather thick, brownish-red mucoid material. This material may contain blood, but often does not. In the latter event it fails to show any of the common reactions for hemoglobin or its derivatives Rigidity is marked and great distention may take place in a short time, so that the liver dullness appears to have been obliterated. Both these symptoms seem to be due to the effect of the violence on the nerve supply. The pulse is rapid, but if the peritoneum has escaped damage, does not become more rapid but gradually becomes slower. Changes in the temperature may or may not take place. Tenderness is most marked at the seat of injury and is limited to this region in the contusions of the wall.

The tenderness and rigidity should not increase to any great degree after the first few hours. The patient should be put at rest in a warm bed and be treated for shock. Opium should not be administered. Progressive muscle spasm and rigidity with increasing tenderness under such circumstances point to involvement of the peritoneum. Changes in the pulse rate, progressing with the local symptoms, indicate the presence of peritonitis. An increasing leukocytosis has the same significance.

Ruptured Viscus.—Hemorrhage from a ruptured viscus will be indicated by pallor, coldness of the skin and a thin, rapid and easily compressible pulse. In cases of hemorrhage rigidity is prominent at once. The endothelium reacts vigorously to the presence of blood and pours out a considerable exudate. Signs of fluid in the flanks, therefore, are likely to be present. Gases free in the cavity lead to the disappearance of liver dullness from the mix-axillary line and prove rupture of the intestine.

INFECTIONS OF THE ABDOMINAL WALL.—If this produces abscess or cellulitis, it may simulate peritonitis, but usually these conditions are distinguished by the more superficial character of the physical signs. The condition is more likely to be confused with a localized peritonitis.

ACUTE CONGESTION OF THE OVARY OR TESTICLE, TORSION OF THE SPERMATIC CORD, ORCHITIS IN AN UNDESCENDED TESTICLE.—These all give rise to abdominal pain and more or less collapse. Local tenderness out of proportion to the general abdominal reaction nearly always makes the diagnosis.

DISTENTION OF THE URINARY BLADDER.—This has been confused with a localized peritonitis. The characteristic shape of the full bladder and the immediate disappearance of the symptoms when it is emptied, either spontaneously or with the catheter, settles the diagnosis.

TUBERCULOSIS OF THE SPINE (POTT'S DISEASE).—This frequently gives rise to pain referred to the abdomen. The onset of the pain may be sudden, but it is rare to find a corresponding degree of tenderness or muscle spasm. Rigidity of the spine precedes the involvement of nerve roots in spondylitis. Similar pains may result from violent twisting or other traumatism of the vertebral column. These referred pains are no doubt responsible for some of the cases of alleged traumatic appendicitis, and for the cases of abdominal lesions said to have been cured by manipulative methods of treatment.

ADRENAL DISEASE.—This condition may be characterized by sudden attacks of abdominal pain with vomiting and collapse. The abdomen is soft and retracted and signs of serious damage the to circulation are present.

MENINGITIS.—This may give rise to abdominal pain with nausea and vomiting and rigidity of the muscles. The muscular retraction is fixed instead of spasmodic, and, like the pain, is caused by irritation of the spinal roots by the exudate. Tenderness is superficial or absent.

ACUTE APPENDICITIS, SALPINGITIS, CHOLECYSTITIS, PANCREATITIS AND PHLEGMONOUS GASTRITIS, RUPTURE OF AN ECTOPIC GESTATION, ABDOMINAL CYST.—These conditions all have many symptoms of their initial stages in common, and these are practically identical with those of the onset of peritonitis. As the particular disease progresses, secondary symptoms develop which identify the source of the trouble. The same is true of cases in which a pedunculated cyst or solid tumor becomes twisted on its pedicle, so that the blood supply is disturbed. In all these conditions peritonitis of some degree either develops with the progress of the original disease or as sequence to it. All these lesions, therefore, require operation for their cure. The best results are obtained when the operation is performed early, while the pathological process remains confined to the organ in which it orignated. The same is true of the early stage of peritonitis. In suspected cases time should not be wasted in an attempt to make an accurate diagnosis beyond the recognition of the crisis which demands immediate intervention.

Acute Intestinal Obstruction.—The differentiation between peritonitis and intestinal obstruction, except at their onset, is usually difficult and often impossible. Laboratory examinations give little help and clinical signs are likely to be confusing. A local peritonitis by infiltration and angulation of the bowel wall can set up an obstruction. The fulminating type of peritonitis causes a paralytic distention of the intestine with stoppage of its contents. A tightly strangulated bowel will allow the passage of infectious material through its damaged wall and thus set up a peritonitis. At the onset the continous pain of peritonitis contrast with the paroxysms of obstruction. Muscle spasm and rigidity of the abdominal wall are prominent symptoms in peritonitis and are absent in obstruction. The tenderness of peritonitis is replaced by a feeling of soreness in obstruction. Fever is more likely to be present in peritonitis and peristalsis is lessened. The development of distention in a quiet abdomen indicates peritonitis and makes a contrast to the noisy abdomen of obstruction with its visible peristalsis and palpable distended coils. The progressive rise in pulse rate is characteristic of peritonitis. It is possible that a rapidly increasing quantity of non-proteid nitrogen in the blood may be characteristic of obstruction rather than of peritonitis.

Hernia.—Strangulated hernia gives rise to pain and tenderness at the point of constriction of the contents of the sac. When bowel is involved, the symptoms of obstruction are added to the local signs.

Internal hernia is seldom diagnosed before the abdomen has been opened. External hernia usually is easily recognized by the presence of the hernial swelling. Small protrusions, especially small femoral hernia occurring in fat patients, may be overlooked, unless the region of the ring is studied with care.

Acute Suppurative Nephritis.—In the early stages of acute hematogenous infection of the kidney the general symptoms of severe infection may simulate those of peritonitis and overshadow those of the unilateral kidney lesion. Spasm of the lumbar muscles with tenderness at the costovertebral border of the last rib gives the clue to the kidney disease. Careful urological study will confirm the diagnosis. Hydronephrosis also may simulate peritonitis. The intermittent nature of the signs and symptoms, the absence of fever and unchanged pulse rate distinguish the hydronephrosis.

Treatment of Acute Peritonitis.—In their early stages cases of perforative peritonitis should be treated **surgically** and those of hematogenous origin by **nonoperative methods.** Too much stress can not be laid on the urgent need for prompt operation in all perforative cases as soon as the patient can be brought into the right environment, which means practically always a properly equipped hospital. The relation of delay to fatal outcome is direct.

A clear distinction must be made between the lesions causing peritonitis and the peritonitis itself. The causative lesions all require surgical operation for their cure and in most cases prompt operation is the only means by which peritonitis can be prevented. This is especially true in appendicitis where misapplied medical treatment of peritonitis has been responsible for delay and its consequent disasters.

Surgery in perforative peritonitis is limited to the following:

(1) Closure of the perforation by the simplest possible means that will insure against leakage. In the gastro-intestinal tract provision must also be made for the flow of material through or around the region of the perforation.

(2) Relief of tension in the infected area. This is accomplished by adequate drainage. Removal of pressure is essential for diminishing absorption from the infected area.

(3) Well-conducted postoperative treatment. This includes the methods discussed in the medical treatment.

No special effort is made to remove the exudate. The intestines are not allowed to escape from the incision and with the other intraabdominal organs are disturbed as little as possible. Irrigation or the introduction of antiseptics does not seem to do good and may do great harm. Crisler of Memphis has had an experience with iodin in peritonitis that is unique.

In peritonitis there is always an increased absorption in the early stage when the endothelial cells bear the brunt of the pathological process and are damaged by mechanical or chemical irritants. Later, when the subperitoneal tissues have reacted to the infection, absorption is diminished by the presence of the wall of granulation tissue so formed.

The number of hours required for the passage from one to the other of these stages naturally varies with the relative resistence of the patient and the virulence of the infection. The quantity of material which escapes into the cavity, as well as the rapidity with which it is poured out, has a decided influence on the degree of virulence. The position of the perforation is a factor.

Material introduced through an appendicial perforation seems to have the greatest bacterial activity. The location of the peritonitis has an important bearing on its course. Pelvic peritonitis is relatively mild, that occurring in the region of the diaphragm severe, with variations as the process spreads along the intervening area. A considerable degree of diffuse peritonitis may exist in the central position of the cavity with relatively mild symptoms. In these cases agglutination of the coils of small intestine limits the process to what is really a small area.

The ideal time for operation is during the first six hours, while the endothelium is still capable of resistance. Removal of the cause of infection at this stage practically always is followed by recovery from the peritonitis. For a varying period after this ideal time, usually from twelve to twenty-four hours after the onset of symptoms, operation will still offer the best chance for speedy recovery. Later than this, when the peritonitis is spreading, operations may do harm by opening up new fields for infection, or otherwise adding to the strain on the patient's vital processes. During this second stage the reactions within the body are making every effort to confine the invasion of the peritoneum and to prevent absorption of septic products into the circulation. The best way to assist nature at this phase of the struggle appears to be by means of the methods discussed as the medical treatment. If this form of treatment proves successful, limitation of the infectious process will give the surgeon opportunity for one or more interventions during the third stage of localized abscess.

Operation during the second stage may become necessary. The determination of this necessity and of the character of the operation to be performed requires good judgment on the part of the surgeon. Cases of appendicial peritonitis as a rule may be operated on successfully during this stage, probably because even large peri-appendiceal exudates are usually circumscribed by adhesions.

In deciding against operation, symptoms of grave toxemia, especially those indicating exhaustion of the vasomotor and respiratory mechanism, are important. A very rapid pulse with relatively low blood-pressure, cynosis or jaundice, or a combination of both discolorations of the skin are bad symptoms. Marked distention with little or no peristaltic movement, dark vomitus and hiccough, great restlessness and a general condition of collapse are contra-indications to operation.

The **medical treatment** of peritonitis attempts to overcome the dangerous effects of infection by assisting the patient in his local efforts to limit extension and absorption, as well as in providing means for relief of the septicemia. The valuable work of Murphy, of Ochsner, and of Crile, is responsible in large measure for the existence of a systematic and well-founded method of procedure in the treatment of peritonitis.

GENERAL CONSIDERATIONS.—The reparative power of the body is augmented by **complete rest.** The patient with peritonitis must be **kept quiet in bed.** He should not be allowed to get up for any purpose and every avoidable activity should be eliminated. Bathing, urination, defecation should be performed in bed with a minimum of exertion. Usually the patient is so ill that there is little difficulty in controlling him.

The room should be **well ventilated** without exposing the patient to cold. Respiratory action becomes limited in peritonitis and therefore an abundant supply of fresh air is a necessity. The surroundings should be quiet and **the number of visitors carefully controlled.**

SPECIFIC INDICATIONS.—Peristalsis is the most important agent in the spread of peritonitis. Absorption is most rapid from the diaphragmatic area, slowest from the pelvic region. The position of the exudate is influenced by gravity and the adhesions. As a result of vomiting there is loss of a great amount of water. Toxins and bacteria in the blood damage the excretory organs and exhaust the vital functions.

These indications are met by the following procedures:

Abstinence from Food.—The normal stimulus to peristalsis is the presence of food in the gastro-intestinal tract. Therefore, **no food of any kind** is given by mouth. Water should not be given if vomiting is a prominent symptom. When the stomach is quiet a **very small quantity of water** may be allowed at intervals. Patients are more often content to do without food than without water. The mouth should be kept moistened with water, or with a mixture of **glycerin, lemon juice and water.**

Lavage.—Vomiting is usually caused by a reversed peristalsis and therefore becomes an evidence of spreading of infectious material. The **stomach tube** is the only means we have for combating this symptom. Lavage brings away the contained toxic material and empties the distended organ, giving relief to both general and local symptoms. The washings should be repeated as often as distention and nausea indicate a filling of the stomach. The tube must be passed gently. **Spraying a two per cent. solution of cocain** on the irritated pharynx makes the introduction of the tube easier. If violent straining and resistance is produced by passing the tube, greater harm is done by spreading infection than good is accomplished by the lavage.

Continued lavage by means of a modified tube devised by Kanavel[8] will do away with the necessity for frequent passage of the stomach tube. In operative cases the tube may be inserted while the patient is anesthetized. This tube resembles the Rehfuss and Einhorn tubes in size and therefore is easily retained in the stomach. It is provided with a stylet to facilitate its passage and an extension for syphonage. The stomach contents are aspirated every hour and the stomach is washed out at eight-hour intervals. Between washings the distal end of the tube is kept under water in a container on the floor so that continuous syphonage takes place.

Posture.—The **upright position,** almost universally known as the Fowler position, is of value because of the effect of posture on the localization of peritoneal exudates and absorption. It is secured easily by using a special form of bed (the Gatch bed) in which the spring is hinged to make three segments. When the special bed is not available, the position may still be obtained by using the ordinary back rest, or one extemporized by putting a straight backed chair upside down in the bed and padding it with pillows. The patient is held against the back rest by a bolster or a roll of blanket placed under the flexed thighs and secured to the head of the bed by a folded sheet or strong bandage. The pillows should be placed so as to support the dorsal spine in the normal lordosis, but should not be thick behind the sacrum, where such an arrangement will flex the back in the lumbosacral region and lead to accumulations in the iliac fossæ rather than in the pelvis. This sitting-up position not only influences the position of the exudate by the force of gravity, but also improves the respiratory movements and thus increases oxygenation.

Water.—**Water** is essential for vital activities. The activity of each cell of the body requires for its maintenance a plentiful supply of fresh water. In peritonitis vomiting removes a quantity of water from the organism, more is lost through the sweating, and much remains in the distended bowels. Because the function of the gastrointestinal tract is deranged to a serious extent, the normal routes of absorption are lost. Water therefore, must be administered by rectum (**proctoclysis**), subcutaneously (**hypodermoclysis**) or intravenously. The simplest method is to administer the water by way of the rectum in the manner described and advised by Murphy.

*Proctoclysis**—Many varieties of apparatus have been devised for proctoclysis. Some of them have advantages, and when available make the rectal administration of fluid easier. Elaborate apparatus however is not necessary. What is required is some sort of reservoir to hold the water, a catheter of about No. 16 (French) size, and a tube and connections to join the two. A graduated glass reservoir makes the rate of flow visible, and some form of warmer will keep the liquid at a reasonably uniform temperature.

Good results however, can be obtained by using a fountain syringe for the reservoir and its vaginal douche-tip for insertion into the rectum. The temperature of the water may be kept at approximately 100° F. by surrounding the tube or the reservoir with hot water bottles. The reservoir should be suspended from 6 to 14 inches above the level of the buttocks, just high enough to allow the liquid to flow out at the rate of about a teaspoonful a minute. Absorption from the rectum ordinarily will take place at about the same rate, so that there will be no accumulation and, therefore, no expulsion of the fluid. The slight elevation of the reservoir permits an easy back-flow whenever the patient passes flatus or strains. In some of the special forms of apparatus devised for proctoclysis arrangements are made for the outgo or flatus and for control of the rate of ingress of fluid by transparent bulbs inserted in the course of the connecting tube. The **quantity** of **water** that it is possible to administer in this way may be very great, and usually reaches **3** or **4 quarts** during the first twenty-four hours. After this time the rate of absorption will be slower, and, as a rule, the rectum expels the fluid after thirty-six hours of continuous administration. At this stage the proctoclysis should be interrupted for a few hours after which the power of absorption by the rectum will be restored. An irritable rectum will not tolerate proctoclysis. In some cases a preliminary starch enema will allay the irritation.

The character of the fluid administered varies. Ordinary drinking water is efficacious and is easily absorbed. The addition of two ounces of **glucose** to each pint of water supplies nutritive material which is of value in combatting the tendency to acidosis present in peritonitis as in all severe infections. The addition of **bicarbonate** of **soda** to the glucose solution has been advised for the relief of the acidosis. There seem to be some physiological objections to the administration of alkalies in cases where the alakali reserve is low. Practically the addition of the bicarbonate to the fluid for proctoclysis appears to increase the distention of the rectum.

Hypodermoclysis.—**Subcutaneous infusion** is more certain than proctoclysis for introducing water into the system. The method requires the use of sterile solutions, aseptic methods of procedure, and a certain amount of special apparatus. Therefore, it is less often available than proctoclysis as a household measure. However the certainty with which the fluid is introduced by hypodermoclysis should lead to the use of this method whenever possible.

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*The Murphy proctoclysis outfit is relatively simple and very efficient. The visible dropper with an outlet for gasses is a useful part of the outfit.

The apparatus consists of a special form of bottle graduated to 800 or 1000 c.c., provided with half inch rubber tubing, to which is attached by appropriate means a hypodermic needle about three inches long and having a bore of approximately 1-32 of an inch (Number 20). Near the bottle the rubber tubing is divided and one of the visible droppers inserted to join the two segments. A screw clamp is put on the proximal segment of rubber tubing to control the flow from the bottle. The entire apparatus may be sterilized by boiling before use. As a rule all of it except the needle is sterilized in an autoclave and kept ready, bundled up in a cover. When sterilized in this latter way some precaution must be taken to prevent angulation and flattening of the rubber tubing. Tubing so damaged in the sterilizer kinks and twists so much that a proper flow does not take place when the apparatus is used.

The fluid administered is heated to about 110° F. (43.33° C.) and poured into the bottle warmed to the same temperature. The bottle is then suspended on an adjustable support and the sterile needle attached to the distal end of the tubing. The needle is passed through a 3-inch square of sterile gauze, 4 to 8-ply thick, which makes a shield at its hilt, to protect the needle from contamination by the hand.

The skin of the patient under and to the outer side of the breast is disinfected by painting the area with tincture of iodin. The clamp is opened until all air is expelled and the fluid flows freely. The rubber tubing is then pinched tightly, and the needle is inserted up to its flange into the areolar tissue under the breast. The point enters near the outer border of the pectoralis major, about the level of the nipple, and the needle is directed upward. A strip of adhesive plaster is placed over the hilt of the needle and the rubber tubing to hold the needle and the gauze shield in place.

Hot water bags are placed over the course of the needle and in the axilla. The rate of flow from the bottle should be regulated by the screw-clamp according to the speed with which absorption takes place.

The tissues receiving the infusion should remain soft and pliable and although edematous, should never be allowed to become indurated. Given slowly, a great quantity of fluid can be administered at one point. The effect of large quantities on the circulation, especially on the heart, must be watched. Any evidence of dilatation of the heart is a signal for stopping the infusion. Suppression of urine, when present, will soon lead to water logging.

The fluids given by **hypodermoclysis** in cases of peritonitis are:

(1) Sterile, freshly distilled water. At the rate of from 40 to 80 drops a minute water may be given hypodermically continuously for several days. The total quantity absorbed may reach 3 or 4 litres a day. There seems to be little danger of giving too much distilled water.

(2) One-sixteenth per cent. solution procain in sterile distilled water will make the infusion painless. As far as can be judged from the

experience of many observers, led by Willard Bartlett, this solution can be administered in any quantity without fear of toxic symptoms.

(3) Sterile 3 per cent. solution of glucose provides material for nutrition and for combatting acidosis.

Intravenous medication has many advantages, but the introduction of any considerable quantity of fluid into the circulation is not without danger. Variations in the concentration of the solutions employed and many other factors require careful consideration. Moreover, the probable effect on the heart of the introduction of the fluid cannot be lightly dismissed. When dealing with a patient who is seriously sick with peritonitis the administration of an intravenous infusion is a matter of considerable risk.

External Heat.—The application of **hot moist packs** to the surface of the abdomen gives comfort and stimulates the circulation and increases the local resistance. Rubbing a small quantity of **mercurial ointment** into the skin of the abdomen during the period of using the packs has been found useful in the Mayo Clinic. The packs should be changed frequently. It is not necessary to add any counterirritant, such as turpentine. These irritants are likely to blister the skin. When the moist, hot pack is unpleasant or difficult to maintain, the **Japanese stove** or an **electric heating pad** will be found useful.

DRUGS.—**Opium** is the one important drug in the treatment of peritonitis. It must never be given before the diagnosis of peritonitis has been made with certainty and the plan of treatment decided upon. The use of opium in appendicitis is improper, because by masking the symptoms, the drug will lead to a false sense of security which is likely to end in the disaster of a perforative peritonitis.

When the peritonitis has been established and a decision made as to the plan of treatment to be employed, opium becomes a valuable agent. The drug slows peristalsis; it relieves pain, and relaxes spasm, thereby aiding the respiratory rhythm; it improves the action of the heart. Inhibiting the activities of the higher centers it insulates the brain from the conflict going on in the abdomen. It gives the patient sleep through which alone he is able to restore the energy of his exhausted vital centers.

Opium is best given by **hypodermic injections** of **morphia.** The initial dose naturally varies with the age, size and general condition of the patient. For an ordinarily robust adult, **¼ of a grain (.0162)** should be given and repeated every half hour until the respiratory rate is brought down to 12 or 14 a minute. The morphia is then repeated at sufficient intervals to keep the respirations in the neighborhood of 14 a minute. The development of cyanosis is the only indication for witholding the drug. The patient is kept narcotized until evidences of recession of the peritonitis become manifest. Opium is not useful in streptococcus peritonitis.

With patients who object to the hypodermic injections, **opium** or morphia may be given by means of **suppositories.** Oral administration is rendered useless by the disturbed function of the stomach.

Purgatives.—These are mentioned, but to be condemned. A purgative which acts does all the things which we wish to avoid in the treatment of peritonitis or its causes. The uselessness of purgatives is shown by the fulminating course of those cases of acute peritonitis associated with diarrhœa.

There is a place for the discriminating use of a purgative in the postoperative treatment of some of the causes of peritonitis, notably appendicitis; but these are not cases of peritonitis, and the purgative is not administered until the diseased appendix has been removed. This judicious use of purgatives after operation is often misnamed the aperient treatment of peritonitis, and therefore has been misapplied with many fatal results.

The profession has learned the danger of opium as a masker of symptoms in peritonitis. It would be life saving, if the greater danger of purgatives were as well understood. Every surgeon has met with cases in which a gangrenous appendix or a strangulated bowel has ruptured as a result of violent purgation, and where a localized peritonitis has been spread wide over the cavity by the vigorous peristalsis set up by similar medication. We have found castor oil floating on the exudate of a ruptured duodenal ulcer, and croton oil in the peritoneal fluid around a leaking cancer of the colon.

Enemas.—Ordinarily in peritonitis, no attempt should be made to move the bowels. When the distention is great enough to cause anxiety, and it is not relieved by **lavage** of the stomach, a **high enema** should be given. Various mixtures have been advised for use in such an enema, which should be introduced gently and at a temperature of about 120° F. (48.880 C.). Half an ounce of dried alum to a quart of water, a pint each of milk and molasses, half a teaspoonful of turpentine, and four ounces of sweet oil with a quart of hot soap suds are among the formulas recommended. A **quart of hot soap suds** is a very useful enema.

Stimulants.—Generally speaking, stimulants are of no value in peritonitis and may do harm. If we accept Crile's theory of the activities of the body, the uselessness of stimulants becomes apparent. In peritonitis a tremendous amount of work is required of the driving mechanism of the organism. As a result the cells become exhausted. Comparing these cells with those of a battery weakened by continuous discharge, we see at once that restoration can not be brought about by forcing the cells to do more work.

Digitalis by its action on the heart has a place in the treatment of peritonitis. It is best given hypodermically as **digitalin,** in doses of from 1/100 to 1/25 of a grain (.00065 to .00259 gram), at four-hour intervals until its influence is noted on the pulse.

Alcohol may be given with the rectal drip. It is of value in severe cases, for example, in cases of typhoid perforation, in the aged and in the case of individuals who have been accustomed to its use in health.

Serums and Vaccines.—These agents have not as yet proved of any value in the treatment of peritonitis.

PROPHYLAXIS.—**Prompt, aseptic surgery** in the treatment of the intra-abdominal causes of peritonitis is the only prophylactic agency whose value is undisputed. Various substances have been injected or otherwise introduced into the peritoneum for the purpose of increasing the local resistance to infection. Practically all of them produce a local leukocytosis with engorgement of the lymphatics. Among the materials used may be mentioned, normal saline solution, horse serum and nucleinate of sodium.

Complications and Sequelae.—ILEUS.—Paralytic ileus may be looked on as a feature in the course of peritonitis. Unless the distention becomes great enough to interfere seriously with respiration, it need not cause alarm. The greatest meteorism is found in the colon. When it becomes necessary to empty the bowel a **high enema,** as a rule, gives relief. When a large area of bowel has been paralyzed it may become necessary to do an enterostomy as described below. Patients who have such severe peritonitis seldom recover.

SPASTIC ILEUS.—Occasionally a loop of bowel, instead of being paralyzed by the toxins of peritonitis, will become spastic from tetanic contracture of the musculature. The intestine is obstructed at the spastic area with resulting distention and toxæmia. The distention here is more likely to be asymmetrical and localized, rather than the general even distention of paralytic ileus, and peristaltic movements will be visible or audible. In such cases **atropin,** given hypodermically in doses of 1/100 grain (.00065 gram) every hour until its effect on the pupil begins to be manifest, will relax the spasm and relieve the symptoms.

MECHANICAL ILEUS.—In the course of peritonitis this is an extremely dangerous complication, whose development it may be most difficult to recognize. Persistent vomiting, not relieved by lavage, intermittent exacerbations of pain and visible or audible evidences of peristalsis are the typical symptoms. Persistence of these symptoms after the effectual use of the stomach tube and enemas will make the diagnosis sufficiently certain to justify enterostomy.

In postoperative cases the original incision may be opened up and a distended loop of bowel, which shows distinct vitality, be brought up into the wound, and there opened and drained. Occasionally it may be more expedient to open the abdomen by a new incision. The method of performing enterostomy described by J. W. Long is most useful.

ACUTE DILATATION OF THE STOMACH.—This complication is most often met with in the postoperative management of peritonitis. The patient becomes distressed, sometimes collapsed. There is great epigastric distention with pain in the region of the nipple. The heart is displaced. There is usually frequent regurgitation of small quantities of dark fluid, which irritates the mouth and chin. Passage of the **stomach tube** allows the escape of a quantity of gas followed by a flow of dark-colored material. Hot water should be poured in through the tube and syphoned off until the washings return clear. Sudden deep discoloration of the washings after they have become nearly clear is an indication of the presence of ileus.

Thoracic Diseases.—Complicating thoracic diseases occur as a rule after the peritonitis has reached its height or has subsided. Embolic pneumonia may be an exception and occur with the spreading peritonitis. Septic pneumonia caused by aspiration of stomach contents during operation in peritonitis may be avoided by operating under local anesthesia, and by thorough lavage of the stomach before the administration of a general anæsthetic.

Suppurating Pleurisy and Suppurating Pericarditis.—These are most often the result of lymphatic extension through the diaphragm. It is obvious that these lesions are more common after cases of peritonitis involving the upper segments of the abdomen.

Phlebitis.—Acute inflammation with thrombosis may occur in the saphenous or iliac veins and extend to involve the vena cava. Thrombophlebitis of the mesenteric veins, and of the portal vein, may lead to abscess of the liver. The thrombophlebitis may give rise to a local abscess, or by means of emboli set up suppuration in the lung or in the brain or meninges. A general pyæmic process may occur.

Kidney Disease.—The kidneys may be the seat of pyæmic abscess or the toxins and bacteria may set up an acute inflammation with suppression of urine. The acute suppression is a complication of the early spreading peritonitis and is a bad omen. Stuart McGuire has found **spartein sulphate** given hypodermically in doses of 1 to 2 grains, (.065 to .13 gram) every three to six hours, of value in some cases of acute suppression.

Urinary Retention.—The bladder may not be emptied because of the general apathy, or because of pain produced by voluntary efforts to avoid urine. The retention may be due to involvement of the bladder wall in the inflammatory process.

Fecal Fistula.—Fecal fistulæ occurring after operation in peritonitis usually close spontaneously or by means of a simple operation, provided there is no obstruction of the bowel beyond the fistula. The exception to the rule is the fistula of tuberculous peritonitis. This cannot be closed and usually leads rapidly to a fatal termination of the disease. Treatment of the ordinary fistula consists in keeping it clean by means of irrigation with a large quantity of sterile water or salt solution once or twice a day, and daily evacuation of the distal segment of the bowel by enemas.

LOCALIZED ABSCESSES

Encapsulation of bacteria and exudate to form a walled-off abscess is the principal method by which the peritoneum overcomes infection. As a rule a solitary abscess forms in the immediate vicinity of the portal of entry, but occasionally an area of suppuration becomes shut off by adhesions to form an abscess in some other locality. The position of such abscesses is determined in part by the various fossæ of the abdomen, and in part by the fortuitous influence of adhesions.

Circumscribed peritonitis may be the result of encapsulation and subsidence of the diffuse variety, or as it often is, may be nonsuppurative and limited from its onset. In the latter type of cases the peritonitis is caused by extention of inflammation from underlying viscera. The process is a relatively dry one, and when it is situated in the region of the diaphragm a friction rub may be heard. The adhesions formed in the nonsuppurative type are usually dense and remain as permanent deformities of the peritoneum, in marked contrast to the rapidly disappearing adhesions of septic peritonitis.

Residual Abscess.—The common positions for residual abscesses are the pelvis, the pericolic region and kidney fossæ, and the subdiaphragmatic space.

The presence of a residual abscess should be suspected whenever septic symptoms persist after a decided improvement in the general abdominal condition has become manifest. Recurrence of fever and tachycardia with other signs of infection after the peritonitis has shown definite abatement practically always indicates the presence of a residual abscess. Considerable time may elapse between the subsidence of the peritonitis and the development of the symptoms of an abscess.

The new process may be ushered in by a chill and high fever with local pain, or the onset may be insidious with the gradual development of hectic fever, with prolapse, sweating, wasting and general debility. The treatment of all these abscesses is by incision and drainage.

Pelvic Abscess.—Pelvic abscess may be residual after diffuse peritonitis, or be a local development from a salpingitis, or after puerperal infection. The pelvic swelling is identified by rectal and vaginal examinations. Prostatic inflammation and distention of the bladder should not be confused with pelvic abscess.

Kidney Fossa Abscess.—Abscess in one or both kidney fossæ may exist, but it is commonest to find the suppuration a pericolic abscess in the right lumbar region. A thick and tender mass is felt in the loin close under the skin. With the general symptoms of infection this mass is not likely to be confused with tumor or inflammation of the colon, the lesions which simulate it. Pus from the right pericolic abscess may extend into the kidney fossa and from there up over the liver or through the foramen of Winslow into the lesser cavity.

Subphrenic Abscess.—Subphrenic abscess may occupy any portion of the space between the diaphragm and the organs in the upper zone of the abdomen. According to their position Barnard divided subphrenic abscesses into six forms. Two of these are extraperitoneal, situated on the right and left sides of the posterior portion of the space where the liver is not covered by peritoneum. The remaining four, are right and left, anterior or posterior, according to the relation of the abscess cavity to the ligaments of the liver. The left posterior intraperitoneal abscess is one confined to the lesser peritoneum. The right and left anterior intraperitoneal abscesses are the commonest varieties.

Unless the abscess is opened and drained extension from the peritoneum will occur. The infection extends into the pleura, lung or bronchus. It may invade the pericardium and has even reached the tissues of the neck. Some of these abscesses may reach the skin at the ensiform, the umbilicus, or between the lower ribs.

The diagnosis of these abscesses is difficult. Pleurisy with effusion, or empyema is a frequent complication; and the subphrenic abscess may or may not contain gas, so that differentiation between thoracic and abdominal lesions may become practically impossible.

Subphrenic abscess may result from direct infection of the lesser peritoneal cavity by perforation of an ulcer of the stomach, or colon, and by extention of infection from the pancreas or spleen. That portion of the subphrenic space which is in the greater peritoneal cavity may be involved as a result of infection of the liver and gall-bladder, or because of extention of suppurative processes from other areas of the peritoneum. About one-half of the recorded cases of subphrenic abcess originated in neglected appendicitis.

Pus may reach the anterior spaces by direct extention over the surface of the liver from an abscess in the right kidney fossa, or it may extend from the pelvis up the left side to form an abscess to the left of the liver, between it and the attachment of the splenic flexure of the colon and the spleen to the diaphragm. The stomach is below the abscess which passes backward to the region of the left kidney.

The local symptoms vary with the position of the abscess, and with the presence or absence of gas in the exudate. Gas is present in nearly one-half of the cases.

When the abscess is situated in the lesser peritoneal cavity there is a smooth tumor in the epigastrium to the left of the middle line, which is behind the stomach and above the colon. The outlines of the tumor as defined by percussion and palpation are changed according as the stomach is full or empty. An extraperitoneal abscess may point just below the ensiform cartilage.

The right and left anterior abscesses give the greatest difficulty in diagnosis, because of the similarity of their symptoms with those of thoracic lesions. The frequent occurrence of a thoracic complication adds to the difficulties. In addition to the general symptoms of residual abscess, the subphrenic type show pain or deep pressure over the involved region. In addition, there may be bulging in the epigastrium or laterally under the ribs. Friction sounds may be heard before the process has lead to effusion into the pleura or pericardium.

The physical signs vary according as the abscess does or does not contain gas, and with the presence or absenee of effusion into the pleural cavity. The presence of a normal area of liver dullness makes the recognition of right-sided abscess more difficult than those situated on the left side.

Right-sided Abscess without Pleurisy and without Gas.—The area of liver dullness is enlarged upward; the edge of the liver may be lower than normal, but as a rule this organ is not pushed down. The level at which the normal respiratory sounds are heard corresponds with upward extention of the dullness. When mapped out on the chest wall the upper border of the dullness shows as a convex line.

Left-sided Abscess without Pleurisy and without Gas.—The area of dullness in the left hypochondrium is easily recognized. The spleen is usually displaced downward and the colon is also displaced downward. The heart may be displaced upward. The upper level of the dullness is convex, and the normal breath sounds are heard at this level.

Abscesses containing gas are more easily recognized. The characteristic sign is the identification of three zones by differences in the percussion note. Above is the normal resonance of the lung; below the dullness of the fluid and the organs making the floor of the abscess; and in the middle, the tympany of the collection of gas in the abscess cavity. On the right side there is little danger of confusion in making out the three zones. On the left side, the possible presence of gastric tympany should be borne in mind. If it be suspected the stomach can be emptied by a tube, or filled with fluid and then identified.

Gaseous abscesses are distinguished from pneumothorax by the fact that the physical signs are limited to the base of the chest and are not present at the apex; and by the absence of lateral displacement of the heart. The heart, if moved at all, is pushed directly upward.

Abscesses with associated pleural effusion depend for their recognition and differentiation upon the history, the mode of onset and course of the disease more than on the physical examination. The area of dullness involves the chest but extends lower into the abdomen than it would with an effusion limited to the pleura. On the left side this change in relations is easily made out, but on the right side the normal area of liver dullness interferes.

X-ray examination is of great value in the diagnosis of subphrenic abscess. Fluoroscopic examination as well as plates should be made whenever possible. The shadow cast by the abscess can be studied in its relation to the diaphragm. Subphrenic abscess raises the diaphragm and its outline is dome-like into the thorax. Pleural effusion depresses the diaphragm and flattens its dome. Suppuration on either side of the diaphragm causes immobilization of this structure. When the subphrenic abscess is complicated by empyema on the same side, there is no way to separate the shadows. The air vesicle of gaseous abscess is shown in the x-ray picture.

Diagnostic Puncture.—When pus is recovered by means of an exploratory aspiration, the diagnosis is usually settled. The distinction between empyema and subphrenic abscess cannot be made by aspiration. Differences in effect of the respiration on the flow of pus, or the movements of the needle cannot be depended on to distinguish between collections above and below the diaphragm. Induration and adhesions about the abscess will separate it from the physical conditions obtaining in the remainder of the pleural cavity.

A negative puncture does not exclude an abscess. The recovery of sterile fluid from the pleura in a suspected case indicates a reaction on the part of the pleura to a neighboring focus and should lead to deeper aspiration.

The deep punctures are not entirely free from danger and may require the use of a general anæsthetic. They are done best in the operating room with preparations made for immediate incision and drainage of the abscess should it be located. The needle should be at least three inches long and of large caliber, so that thick pus may be drawn through it. The aspirating syringe should be of glass and have good suction power. The punctures are made in the scapular and the mid-axillary lines on the suspected side, beginning at the tenth interspace and if necessary, successfully entering the next higher interspace up to the sixth.

SPECIFIC VARIETIES OF PERITONITIS

Streptococcus Peritonitis.—Infection with this organism is generally associated with an overwhelming septicæmia having relatively few signs of reaction or resistance on the part of the peritoneum. The serosa becomes red and brittle, there may be a small exudate of straw-colored, milky or blood-stained fluid. The organism quickly penetrates the subserous layer, giving the endothelial cells little or no opportunity for making a defense. The disease usually runs a rapidly fatal course. When the patient survives the immediate infection of the peritoneum, metastatic abscesses in the liver and the thoracic organs, pyleophlebitis, joint infections, etc., are likely to occur as later manifestations.

The infection may be a postoperative one, or may follow perforation of one of the viscera. Many cases are the result of puerperal infection or criminal abortions. The organism reaches the peritoneum by way of the lymphatics or by escape through the Fallopian tubes; or the involvement may come from rupture of an abscess of the uterine wall, the pelvis or a thrombophlebitis.

Septic sore throat and influenza undoubtedly are the primary foci in certain cases. The infection of the peritoneum in these cases seems most often to be a hematogenous one, although in some cases appendicitis or intestinal ulceration may precede the peritonitis.

Because of the puerperal cases, streptococcus peritonitis occurs most often in women. The cases due to septic sore throat are most commonly found in children.

The outlook in streptococcus peritonitis is very bad. Most patients die. The disease is often overlooked because the local symptoms are relatively mild in comparison with the intense septicemia or the severity of the primary disease. The puerperal variety may be limited to the pelvis, or form localized abscesses, the drainage of which gives good results. The bacteria in these localized collections retain their virulence for a long time, therefore secondary operations after puerperal infection should be done with great care.

In the presence of a streptococcus peritonitis the medical treatment should be carried out. As has been noted, opium is not useful in streptococcus peritonitis. Antistreptococcus sera and vaccines have proved of no value in the treatment of the disease.

Gonorrheal Peritonitis.—The gonococcus almost invariably reaches the peritoneum by way of the Fallopian tube. The extremely rare cases of gonorrheal peritonitis reported as occurring in males probably are really cases of lymphangitis and lymphadenitis along the course of the vas deferens and in the postperitoneal nodes.

The infected tube may set up either of two forms of peritonitis. The commoner variety is limited to the pelvis. Bacteria from the acute salpingitis make their way through the wall of the tube and set up a local peritonitis with a good deal of fibrinous exudate. Coagulation of the exudate and infiltration of the subserous tissues, with agglutination of adjacent coils of intestine limits the spread of the peritonitis.

The resultant mass may be felt as an abdominal swelling coming up from the pelvis and its relations made out by rectal, vaginal or bimanual examination. The mass becomes smaller as the process recedes, which is the natural course of the disease.

Diffuse Gonorrheal Peritonitis.—In the rarer form the peritonitis is diffuse. The bacteria seem to escape from the tube and reach wide areas of peritoneum. This variety may be part of a general infection as is shown by the recovery of the gonococcus from the blood and the occurrence of endocarditis, arthritis and other metastatic lesions.

It is an acute dry plastic form in which there will be found many adhesions with a small quantity of thin greenish exudate containing flakes of fibrin. The disease tends to localize itself about the portal of infection.

The diagnosis of gonorrheal peritonitis is made on the recognition of the primary focus and the mild general symptoms as compared with the intense local ones. The presence of gonococci in the vaginal discharge of a patient having symptoms of peritonitis is suggestive, but not pathognomonic of gonorrheal peritonitis. Gonorrheal vaginal discharges may be present with other forms of peritonitis.

The treatment is expectant, the patient being managed as outlined in the medical treatment of septic peritonitis. Operation is required for removal of residual processes. The bacteria in the residual abscess are rapidly attenuated and the pus soon becomes sterile.

Pneumococcus Peritonitis.—While it is true that perforation or direct extention is responsible for the peritoneal invasion in many cases, it is also certain that pneumococcus peritonitis may occur as a true hematogenous infection. The peritonitis may be the only local evidence of a pneumococcus septicæmia. The disease almost always occurs in children, and in girls about three times as often as in boys. The peritonitis may be diffuse or localized at the onset.

Whether or not the diffuse form may become localized has been disputed, but it seems that localization does occur in favorable instances. The exudate is fibrino-purulent, yellow green in color, odorless, and contains the pneumococcus. The peritoneal reaction is not intense. The surface becomes reddened, the subperitoneal tissue thickened and edematous; the intestines seem to float about in the exudate.

Parker Syms[10] describes three states of the disease:

(1) The stage of toxemia in which the general symptoms of infection are severe and are out of proportion to the signs of abdominal disease.

(2) The stage of peritonitis in which the local signs of a spreading peritonitis become evident.

(3) The stage of recession, in which the peritonitis becomes encysted and forms a localized abscess. The swelling is almost always below the umbilicus and bulges one side of the abdomen. The abscess may point and open spontaneously at the umbilicus. In fatal cases there is no stage of recession, but both the toxic symptoms and those of peritonitis become more intense and pass over into those of fatal general septicæmia.

The symptoms generally begin with a chill followed by high fever, delirium and other evidences of severe intoxication. Diarrhœa is practically always a marked feature of the disease. The abdomen becomes distended and tender, but there is little muscle spasm or rigidity. The abdominal wall is more or less infiltrated and as a result may have doughy feel. The abdominal signs are always less marked than the symptoms of infection during the active course of the disease. Herpes on the lips is almost characteristic of pneumococcus infection.

The organism is present in the blood, but several cultures may be required before it can be made to grow in the test tube. The blood count shows a marked leukocytosis with great increase in the polynuclear leukocytes. The highest blood counts recorded in the laboratory of the Mercy Hospital, Baltimore, are from cases of pneumococcus peritonitis. In one of these the total leukocytes were 53,600 with 98 per cent. of polyneuclears. The diagnostic puncture, recommended by Denzer, might be of value in the diagnosis of the disease and in securing a specimen for the identification of the type of organism present.

Pneumococcus peritonitis should be treated by medical methods until the process becomes localized. The walled-off abscess should be drained. In the diffuse form the use of antipneumococcus serum may add to the probability of cure. If the group one type of organism is known to be the one involved the serum should always be used. In the absence of facilities for identifying the type of organism in the diffuse cases it is reasonable to administer the serum in the hope that the infection is of the type to be benefitted. The possibility of a serum reaction is the only risk and may be disregarded. The serum has been used in only a few cases and in these it has not been of any appreciable benefit.

Tuberculous Peritonitis.—Infection by the tubercle bacillus may cause one of a number of pathological changes in the peritoneal cavity. The variations depend on the quantity of infectious material introduced; the position of the primary focus; the relative virulence of the bacilli and the resistance of the patient.

Many classifications have been made depending on variations in the pathological process and the clinical course of the disease. Like tuberculosis elsewhere the peritoneal disease is versatile in its manifestations and a maze of groupings exist.

The essential lesion, the tubercule is the same in all locations. The tubercule may undergo caseation, or fibrosis; may remain discreet or become confluent; may be surrounded by various degrees of inflammatory reaction; and may grow on the surface of the endothelium, or in the subserous layer. In its growth the process may be associated with an exudate, with the production of adhesions, or with ulceration.

Practically three pathological varieties may be described: (1) Ascitic, (2) adhesive and (3) ulcerative.

The infection of the peritoneum may be hematogenous, or it may come from a focus in the cavity. The common primary foci within the abdomen are the Fallopian tubes, the ileocecal region of the intestine, mesenteric and retroperitoneal lymph glands. Genital tuberculosis in men may lead to peritonitis by extention from the vas, seminal vesicle or prostate. The route by which the bacilli travel to reach the peritoneum is not always clear.

Local irritation may localize the disease, as for example, in the case of tuberculosis of a hernial sac. Trauma to the abdominal wall seems to have set up the peritonitis in certain persons already infected with tuberculosis. A patient having a primary focus of tuberculosis may develop peritonitis after some other disease. This sequence has been noted especially after exanthematous fevers and cirrhosis of the liver.

The distribution of the tubercules shows the influence of gravity and of the peritoneal currents. They are most prominent in the pelvis and in the region of the diaphragm. The omentum is frequently invaded, and helps in the localization of tubercules on the peritoneum of the anterior abdominal wall.

Both the human and the bovine tubercle bacilli are capable of producing peritonitis in human beings. The bovine bacillus forms a pedunculated nodule which hangs from the peritoneal surface by a slender tubular pedicle. The pedicle may become closed and constricted until finally the nodule separatese and forms a free body in the peritoneum, similar to the rice bodies in joints and tendon sheaths.

THE ASCITIC FORM.—Some exudate is found at some stage of the process in all forms of tuberculous peritonitis. The character of the fluid varies. In an acute invasion it may be bloody. A fibrinous exudate precedes the formation of bands in the adhesive type. The quantity of fluid present varies from a few ounces to several quarts. The fluid may be free in the peritoneal cavity, or be sacculated by adhesions. Small cystic tumors may be formed on the wall of the intestine in the region of a tubercular ulcer. These are tuberculous cysts and must not be confused with the sacculated or encysted exudates mentioned above. In the ascitic type the fluid is thin, straw colored and seldom coagulates spontaneously. The tubercules appear to be largely on the endothelial surface.

THE ADHESIVE FORM.—In this type the infection is apparently largely subperitoneal and the progress of the disease is accompanied by a fibrinous exudate which agglutinates adjacent organs and becoming organized binds them together with dense adhesions. The formation of adhesions is a reactive phenomenon. If the tubercules heal, the adhesions loosen and gradually are absorbed. Loose and fibrous adhesions in which only a few encapsulated tubercules are found, indicate a vigorous resistance on the part of the patient.

A vigorous subperitoneal reaction in the region of the primary focus may produce a tumor-like mass of fibrous granulation tissue. In the ileocecal region and along the colon the mass may become large and so indurated that it appears to be carcinomatous. The omentum, when involved, may thicken and roll up to form a tumor-like mass in the upper abdomen.

THE ULCERATIVE TYPE.—As the intestinal wall becomes thinned by a tubercular ulcer, adhesions form about the area. When the ulcer perforates a localized abscess forms in the mass of adhesions, or adjoining coils become united by a fistula. Clinically there is an acute form which is relatively rare, and a subacute and chronic type of the disease.

ACUTE TUBERCULOUS PERITONITIS.—At the onset the symptoms may be predominantly abdominal, or they may be the systemic evidences of acute infection. In the abdominal cases pain, vomiting, and distention with fever and rapid pulse make the onset difficult to distinguish from that of acute perforative peritonitis. As a rule there is less muscle spasm and tenderness in the tuberculous disease. The pulse rate does not show the continuous increase characteristic of septic peritonitis. The blood pressure is low.

Leukopenia rather than leukocytosis is the rule in tuberculous peritonitis. In those cases which show a leukocytosis the increase in the small mononuclears usually is more prominent than that in the polynuclear white cells.

The cases showing systemic symptoms begin with chill, fever and headache, usually accompanied by more or less abdominal distention and colicky pain. Confusion with typhoid fever is common. Recovery of the organism from the blood culture will identify the early stage of typhoid fever, while a positive Widal agglutination reaction is characteristic of the latter stage of this disease. Negative blood cultures or negative Widal reactions do not necessarily prove the absence of typhoid fever. Recovery of the typhoid bacillus from the feces makes the diagnosis of typhoid fever positive.

An exudate occurs early. In the ascitic form the sudden development of fluid in the abdomen is quite characteristic. In children a suddenly developed ascites practically always is due to tuberculosis peritonitis. Bladder symptoms may occur early in the disease. Some gastro-intestinal symptoms are present as a result of adhesions or stiffening produced by a deposit of tubercle on the intestine. Evidence of tuberculosis elsewhere in the body gives a clue to the cause of the abdominal disease.

SUBACUTE AND CHRONIC TUBERCULOUS PERITONITIS.—These forms are much more common than the acute variety. The early symptoms are indefinite and the development of the disease is insiduous. As a rule the first symptoms are those of gastro-intestinal disturbance. Irregular, mild attacks of colic with constipation or diarrhœa, and a soreness of the abdomen are the usual symptoms. Irritability of the bladder is common. As the disease develops the patient becomes anemic and loses weight and strength. The pulse becomes rapid and the blood pressure falls. A slight evening rise of temperature is always suggestive.

The abdominal symptoms vary with the pathological process. Quite early there will be areas of tenderness with slight muscle spasm and rigidity in the location of peritoneal involvement. These signs are never intense, but are notable and persistent. Later on, signs of one or the other pathological forms will predominate.

In the ascitic form the effusion generally becomes noticeable a few weeks after the onset, but may be insignificant in quantity for months. When the fluid is free in the cavity, the association of an evening rise of temperature and some intestinal disturbance will make the diagnosis. The simultaneous presence of effusion in the peritoneum and in the pleura is almost certain evidence of tuberculous disease.

When the effusion is sacculated, it may lead to confusion with other cystic swellings of the abdomen. The most frequent of these is ovarian cyst. As the tuberculous peritonitis so often begins with tuberculous salpingitis, sacculation of the exudate about the primary peritoneal focus gives these collections of fluid similar anatomical relations. They are differentiated by study of the systemic effect of the tuberculosis.

In the adhesive types rounded or flat masses develop, which consist of agglutinated areas of intestine and other abdominal viscera. The intestines are more or less distended, the abdomen is swollen and tympanitic, and there is widespread tenderness and resistance. The nodules may resemble any of the abdominal tumors, and when a considerable area of large intestine is involved, are likely to be confused with cancer. . The tubercular nodules are always more fixed than tumors of the same size. The systemic evidences of infection are more prominent in tuberculosis than in the case of tumors.

In the ulcerative form, the mass of adherent intestines, containing within itself areas of caseation and ulceration, becomes adherent to the parietal peritoneum and forms a tender tumor. At the same time the involved intestine becomes spastic or occluded and signs of obstruction become apparent. Diarrhœa is caused by the intestinal ulcers and may be severe and constant, or alternate with constipation and distention. Fever of the hectic type is common. Perforation of an ulcer into the adherent mass sets up secondary infection and an acute abscess results. This abscess may reach the skin surface and open spontaneously, or it may rupture into the general peritoneal cavity.

When such abscesses involve the liver or spleen, symptoms pointing to these organs will develop. After these abscesses have opened on the skin surface, a fecal fistula forms and is likely to be persistent. Spontaneous opening of the abscess is likely to occur in the umbilical region.

A fecal fistula near the umbillicus is almost certain evidence of the presence of tuberculous peritonitis.

The chronic form of tuberculous peritonitis may resemble almost any chronic intra-abdominal lesion. It is differentiated from confusing lesions by the general symptoms of chronic tuberculosis, that is, fever, weakness and loss of weight. A general and local reaction to hypodermic injection of a diagnostic dose of tuberculin may become necessary for the absolute identification of the disease. Diagnostic puncture and study of the peritoneal exudate may be done, but the procedure is not free from danger. Perforation of an adherent loop of bowel is possible and is likely to cause a fecal fistula.

Treatment.—Rest, good food and the **general hygienic management** of tuberculosis are required in the treatment of the peritoneal type, as well as in any other form of the disease. It is seldom that all the foci present are contained within the abdomen.

Heliotherapy and **x-ray** are especially valuable in tuberculous peritonitis. **Tuberculin** is useful with patients who have slight fever. As with all other methods of treatment of tuberculosis, the best results are obtained when the patient is **in a well-conducted sanitarium.**

Oxygen inflation of the peritoneal cavity has given some excellent results. The puncture must be made with care and any ascitic fluid drawn off before the gas is injected. Repeated inflations may be employed.

Surgical operation is indicated in the early stage of the disease in those cases where it is possible to remove the primary focus. The results of early intervention are brilliant in the cases of peritonitis developing as an acute or subacute process from tuberculous Fallopian tubes. When the primary focus can not be recognized, operation should be delayed, and become an incident in the general management of the patient.

The good effect of laparotomy when the focus cannot be removed seems to depend upon a reactive hyperemia of the peritoneum. The best results have followed operation in the ascitic form of the disease although some improvement has been noted in the adhesive type.

The ulcerative type of the disease will require **operation** for the drainage of localized abscesses. Whenever the abdomen is opened in tuberculous peritonitis, great care is required in the handling of the intestines, especially in separating adhesions. Damage to the bowel will produce a fecal fistula which will resist healing for a long time, and often become permanent. The presence of a fistula interferes with the general nutrition and in this way diminishes the patient's chances for recovery.

Acute intestinal obstruction may complicate either the adhesive or ulcerative variety. This is an especially serious complication. **Prompt operation** may make it possible to separate the obstructing bands without damage to the intestinal wall. Later, a short circuiting operation around the adherent obstructed loops may be the expedient procedure. In late cases an enterostomy becomes a desperate necessity.

Operation for tuberculous peritonitis should not be done in the presence of active tuberculosis elsewhere in the body. It should not be done where there is continuous high temperature. As a result of all operations there is a general reaction like that following an injection of tuberculin, in which the rapid rise in temperature may become alarming. It must be borne in mind that, except in those cases where the primary focus can be removed, operation in tuberculous peritonitis may be followed by an acute miliary development which will prove rapidly fatal.

CHRONIC PERITONITIS

Chronic peritonitis may be localized or diffuse. In some instances the underlying cause may be found, or its nature conjectured from the position and character of the pathological picture. In many cases, especially in the diffuse form, the cause of the disease cannot be determined. Chronic tuberculous processes in which fibrosis has replaced the tubercles may explain some cases. Syphilis is the cause of some others. Leutic peritonitis may be local about a gumma or diffuse.

Actinomycosis of the peritoneum may follow perforation of a nodule from the intestine or one of the other abdominal viscera. W. S. Gardner of Baltimore has reported a case of actinomycosis of the ovary. The peritoneal masses soften and form chronic abscesses, ulcers or sinuses. The pus contains sulphur yellow granules made up of masses of the ray fungus. These granules in the pus are characteristic of actinomycotic infection.

Chronic peritonitis may follow a traumatism of the abdominal wall. Foreign bodies, whether introduced through a wound in the wall or by perforation from the intestine, may set up a chronic reaction. Repeated tappings of an acites can be the cause by producinig an inflammatory reaction possibly by a mild infection. Severe anemias may produce the disease.

Two types are noted. In one, inflammatory adhesions of varying extent and density mark the process; in the other, an ascitic exudate is the prominent characteristic. The ascitic variety is associated with the formation of more or less inflammatory connective tissue and in some cases the exudate seems to follow interference with the intraperitoneal circulation by this inflammatory tissue.

Localized chronic adhesive peritonitis is a familiar condition which occurs at the site of a gradual perforation into the cavity or in the region of a previous acute circumscribed peritonitis. The adhesions may be extremely dense, binding loops of bowel and other viscera into firm masses which may be attached to the parietal peritoneum. On the other hand the adhesions may take the form of web-like veils in various portions of the abdomen. Some of these bands are believed to be of congenital origin.

The character of adhesions seems to vary with the cause of the peritonitis and with the individual type of resistence. With the same etiological factor one individual may develop a few web-like adhesions which cause no symptoms; while another develops firm bands which give rise to all manner of disturbance. It seems reasonable to presume that the tendency to form adhesions after injury to the peritoneum is of the same nature as is the tendency to form keloid after injury to the skin. Patients who have a visceroptosis seem especially prone to develop adhesions after laparotomy.

The diffuse type is rare and is often associated with chronic inflammation of the pleura and pericardium. The association of inflammation of these three serous sacs is known as chronic multiple serositis. There is some discussion regarding the original seat of the disease. The peritonitis may be secondary to the heart lesion or the causative agent may occur primarily in the peritoneum and extend through the diaphragm to the thoracic sacs.

A slowly developing ascites is the usual course of the disease. As a rule the fluid is free, but it may be sacculated. The peritoneum becomes thickened and very white. The liver, spleen and the intestines may be encased by a thick membrane. Kelly named this condition **iced liver.** When the membrane is cut through the normal appearing liver is discovered beneath it.

Miles Porter and Welch report a remarkable case of chronic peritonitis in which the membrane pleated up the intestine like an accordeon and produced a fatal obstruction. In some cases the omentum becomes rolled up into a rounded mass extending across the upper abdomen. The mesentery may become infiltrated and shortened, so that the intestines are held close to the vertebral column and become completely submerged in the exudate.

Symptomatology.—The symptoms of chronis peritonitis vary to a great degree. Adhesions may exist without any symptoms, or they may give rise to pain, to obstructive symptoms, or to reflex disturbances within the abdomen. As a rule the viscero-parietal adhesions are the painful ones. Masses of adherent viscera may resemble any of the benign or malignant tumors. The abdominal wall often feels doughy over the inflammatory masses. When there is a sacculated exudate it may be mistaken for an ovarian cyst; a pancreatic cyst, or a hydronephrosis.

The true cysts are regular in outline, while the sacculated exudates are likely to be nodular. The tumor of hydroneprosis is intermittent in its size. Pyelography will make the identification of the kidney swelling certain.

The general symptoms of chronic peritonitis are a dull feeling of heaviness in the abdomen, associated with ascites, and occasionally the presence of the inflammatory masses. In the diaphragmatic cases there may be pain and tenderness in the lower part of the thorax. The ascites will cause a general dullness on percussion of the abdomen when the intestines are held against the vertebræ by a shortened mesentery.

The urine may show the changes of granular nephritis. Edema of the lower extremities does not occur until the disease has been present a long time. The disease may occur at any age and seems evenly distributed between the sexes. A number of cases have occurred in children, but most of those reported have been in persons of middle life.

Acute or chronic intestinal obstruction is the most serious complication of chronic peritonitis. The treatment of the peritonitis will often be that of the obstruction. Division of bands and the covering of raw surfaces by peritoneum, short circuiting operations around the adherent masses and similar procedures may be required.

Treatment.—When there is a possibility of removing the cause of the peritonitis, for example by closing a chronic perforation, by excising an inflamed area, or by removing a foreign body, operation is indicated. Laparatomy for division of adhesions should be done after judicious consideration. Unless there are definite symptoms of obstruction viscero-visceral adhesions are not likely to be doing harm. The part of adhesions in producing pain must be decided after careful study of the character and distribution of the pain and the probable seat of the adhesions. Much damage has been done by ill-conceived or unnecessary operations for the relief of adhesions.

In chronic diffuse peritonitis with ascites operation is seldom indicated. The fluid should be removed as often as is necessary for the comfort of the patient. When the underlying cause is known this should be treated. For example, luetic cases should receive vigorous anti-luetic medication.

Actinomycosis is treated by intermittent administration of iodid of potash and with copper salts locally applied.

In some cases chronic peritonitis has been found when the abdomen was opened for some other lesion. Many of these patients have recovered from all symptoms after the operation. It may be that the unknown or unrecognized cause of the chronic peritonitis was removed at the operation.

Carcinomatous Peritonitis.—This resembles the tuberculous variety. The disease practically always follows cancer of one of the intra-abdominal viscera. The rare colloid carcinoma will be discussed with the tumors of the peritoneum.

The reaction of the peritoneum to secondary carcinosis usually is associated with an exudate of blood-stained fluid. The cancer metastases form nodular masses in the omentum, and on the parietal and visceral layers. The parietal masses may infiltrate the abdominal wall in the region of the umbillicus. Metastatic enlargement of the glands in the left supraclavicular triangle may give the clue to the cancerous nature of the disease. The inguinal glands may also be involved. Careful examination should be made of the stomach, the pancreas, the colon, the rectum and of the ovaries. These organs are the common primary seats of intra-abdominal cancer. The general clinical picture is that of emaciation and cachexia with ascites.

Carcinomatous peritonitis is evidence of dissemination of cancer. The outlook for the patient therefore is practically hopeless. The relative malignancy of different forms varies within wide limits. In some cases of peritonitis secondary to malignant papillomatous cyst of the ovary the patients have lived many years.

W. J. Mayo (*l. c.*) reports a case of chronic peritonitis operated on two years after a papillomatous cyst had been punctured. At the second operation the sclerosed peritoneum was free from metastases. The cyst and a large collection of ascitic fluid were removed.

TUMORS OF THE PERITONEUM

Tumors of the peritoneum may be either solid or cystic in their structure and benign or malignant in their characteristics. They may grow on the endothelial surface, but are more common in the subserous layer.

Benign tumors are relatively rare. The solid tumors are most often lipomas of the subserous layer, originating in the retroperitoneal space below the level of the renal vessels. Subserous lipomas may occur in the gastro-intestinal tract. These tumors may be globular or nodular, pedunculated or sessile. Like all the retroperitoneal growths, which usually contain more than one type of tissue, the lipoma almost always consists of a considerable quantity of myxomatous material as well as fat. The myxolipoma differs from the pure fatty tumor in containing element of malignancy. Other fatty tumors contain a greater or less quantity of fibrous tissue within their capsule.

The nodular tumors may extend in many directions, some of the lobules may involve a great vessel or an important viscus in their growth by surrounding it, or by adhesions.

The peduculated tumors may be moved about by peristaltic action. In this way the pedicle is stretched and the tumor acquires a wide range of motility. In some cases the pedicle becomes thin and is broken off, the tumor then wandering about the peritoneal cavity until it finds a secondary attachment. Dr. John Thorkelson, some time Professor of Anatomy at the College of Physicians and Surgeons, Baltimore, found one of these wandering tumors free in the peritoneal cavity of a cadaver. The mass was ovoid, about two and one-half inches in the long diameter, encapsulated, and histologically it was fibrolipoma.

Properitoneal lipomas develop in the region of the hernial rings and about the vessels which penetrate the linea alba. As a result of muscular action in their vicinity these fatty tumors are thrust forward so that they make traction on the peritoneum. As a result, a process of parietal peritoneum may be pulled forward to make a hernial sac. This is especially true in the epigastric region, where small hernias of this type are fairly common and produce varied gastro-intestinal symptoms.

The cystic tumors may be the result of embryonal inclusions, for example, the dermoid cysts; or they may be inclusion cysts of later origin, for example, the cysts about encapsulated foreign bodies. Other cysts are formed by degeneration processes in solid tumors or organs. Finally, there are hydatid cysts of the peritoneum, some of which seem to be primary in the serosa, while others are the result of rupture of a hydatid cyst of the liver.

Enterocysts are derived from some abnormality in the development of the intestine. They are almost always found in relation to the omphalomesenteric duct or Meckel's diverticulum. These cysts are distinguished from intestinal diverticula by the fact that they do not communicate with the lumen of the bowel.

Malignant tumors of the peritoneum are more common than the benign ones. They are of two kinds:

(1) Epiblastic, which are secondary growths on the peritoneum.

(2) Mesoblastic, which are less common, and may be either primary or secondary.

The epithelial tumors reach the peritoneum by direct extension from cancer of an abdominal organ, most often the stomach, or the ovary; by lymphatic extension, principally through the mesenteric or postperitoneal glands; or by way of the blood stream. The peritoneal growth may consist of the very soft, gelatinous fungus masses of colloid cancer; the smaller and harder nodules of medullary carcinoma; the medallion like hard plaques of schirrus; or the widespread seed-like granules of miliary carcinomatosis.

The mesodermic tumors of the peritoneum are:

(1) Endothelioma, which arises either from the cells of the serosa, or from those of the lymphatics. In appearance and distribution these tumors resemble cancer of the peritoneum.

(2) Sarcoma are usually large solid tumors which are likely to undergo cystic degeneration.

(3) Lymphadenoma most often involving the retroperitoneal lymph glands.

The clinical symptoms vary with the nature and extent, as well as with the position of the peritoneal tumors. The general symptoms are those of change in health with abdominal pain which may be slight or severe. Alternating diarrhea and constipation is common. A peritoneal exudate with the presence of irregular masses in the abdomen makes the diagnosis almost certain. The masses are larger than those found in tuberculous peritonitis. Tumors of the peritoneum are most common in adults while nodular tuberculosis is more common in children. The ascites may be a prominent symptom and require tapping. Recovery of a hemorrhagic exudate, or one containing endothelial cells and lymphocytes, indicates the presence of tumor.

Pressure of the tumor on the great vessels interferes with the return circulation and produces edema of the legs and scrotum. The colon is carried on the summit of the retroperitoneal tumors towards the middle of the body. Its position and relation may be recognized by inflation of the bowel, or by x-ray pictures.

Intestinal obstruction is a common complication, and the symptoms of obstruction may be the first indication of the presence of the tumor. In other cases the existence of the tumor may be discovered at the operation for the relief of the obstruction.

Pseudomyxoma is a condition in which the peritoneal cavity contains an encysted or free collection of colloid exudate which may be mucilagenous or gelatinous in consistency. The condition is caused by the rupture of a colloid growth of either the ovary or the appendix. The course of the disease may be rapidly fatal, or the patient may recover after removal of the primary focus and part of the exudate. Apparently some patients have recovered spontaneously. These variations in clinical course indicate variations in malignancy of the primary foci.

The symptoms are the general ones of peritoneal tumor, with some local signs of tumor in the region of the primary focus.

Treatment.—Laparotomy should be done as soon as the presence of the tumor is suspected. Many of the benign tumors can be removed with ease. Others may present difficulties on account of adhesions and extensions into inaccessible localities. The malignant tumors almost always are hopeless. Pseudomyxoma should be treated by **removal of the primary focus** and as much of the exudate as can be scooped out easily by the hand, without making any attempt to get rid of all the colloid material. The abdomen should not be drained.

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CHAPTER XXXV

ASCITES, DISEASES OF THE OMENTUM AND MESENTERY

BY A. W. CALLOWAY, M.D.

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ASCITES

Definition.—Ascites has been variously defined by different authors. According to the older definition ascites is a collection of serous fluid in the peritoneal cavity while more recently and correctly it has been called an accumulation of fluid freely movable in the peritoneal sac. Ascites in Greek was known as the "wine skin" disease, owing to the appearance of the abdomen simulating a tight wine-skin. The assumption was that a wine-skin contained either a clear or yellow fluid, and the distention of the abdomen with fluid marked the resemblance. While it is true that most of the fluid accumulated in the peritoneal sac comes about slowly producing the appearance of a distended wine-skin, there are at least two conditions, hemoperitoneum and choleperitoneum, which develop very rapidly, and even though they are the accumulation of free fluid in the peritoneal cavity, they probably do not conform with what was meant by the word ascites. Therefore there appears to be no reason why ascites cannot be clinically studied as an accumulation of fluid in the peritoneal cavity.

From a clinical standpoint it is necessary to make a study of all of the fluids which may be found in the peritoneal cavity, and the etiology of the accumulation of these fluids.

Physiology.—It might be well to review the physiology of this interesting sac, and make a few observations. The extent of the surface of the peritoneum is about the same as that of the external surface of the body. Several investigators attempted to show that certain stomata lead directly into the serous and subserous lymph spaces, thereby establishing communication between the peritoneal cavity and lymphatic system; however, more recent writers do not hold that opinion. The important lymphatic trunks draining the peritoneal cavity are those leading into the anterior mediastinum. Oppel suggested that the lymph stream from the peritoneal cavity is chiefly by the great omentum. There probably are secondary outlets. A small quantity of serous fluid is always present in the peritoneal cavity, and constantly interchanges between the two layers of the peritoneum, the absorption being partly from the lymph vessels and partly directly by the blood stream. In other words it is both a vital and a physical process. Various factors influence the rapidity with which fluid is absorbed; first, the pressure exerted by the abdominal muscles, especially the rhythmical action of the diaphragm; and, second, the peristalsis of the intestine.

Ascites in patients with cirrhosis is due to a number of factors, but a mechanical stasis with toxemia affords the most satisfactory explanation. Re-absorption is retarded by venous engorgement when the tension and pressure is increased. Clinically it does seem that rapid peristalsis promotes absorption, and unless transudation from the peritoneum runs parallel with absorption there must be an accumulation of fluid. In normal conditions this is carefully regulated. Pathologically there is a lack of balance of these factors, relative increase in transudation or relative diminution of absorption resulting in an accumulation of fluid.

Etiology.—The conditions causing fluid in the peritoneal sac, arranged in order of frequency are as follows: (*a*) Cardiac weakness impairing the myocardium; (*b*) renal diseases impairing the kidney function; (*c*) hepatic cirrhosis; (*d*) peritoneal tuberculosis; (*e*) intestinal obstruction; (*f*) abdominal tumors (both malignant and benign); (*g*) abdominal cancer in various localities; (*h*) uterine fibromyoma; (*i*) peritoneal carcinosis; (*j*) pericardial adhesions; (*k*) hepatic carcinoma; (*l*) pernicious anemia; (*m*) leukemia; (*n*) mesenteric thrombosis; (*o*) abdominal lymphorrhea; (*p*) viceral syphilis; (*q*) caval and portal thrombosis; (*r*) large pleural effusion; (*s*) tumors of the abdomen, and pressure on the vena cava or portal vein; (*t*) tumors of the kidney, sarcoma, hypernephroma and hydronephrosis; (*u*) perisplenitis; (*v*) periphepatitis; (*w*) peritoneal adhesions, binding the portal vein as it enters the liver; (*x*) ankylostomiasis; and (*y*) echinococcus.

The most exhaustive etiological study has been made by Cabot, from a clinical report of 3,074 cases, and a report of autopsy of 224 cases. These reports show that heart and kidney disease, abdominal neoplasm, cirrhosis of the liver and tuberculous peritonitis caused about 88 per cent. of all ascites. He mentions eclampsia as a causative factor or related in three cases. Of unusual interest was the high percentage of ascites in ovarian cysts and tumors, as well as the large number found in intestinal obstruction.

Symptomatology.—Whether the fluid is of rapid or slow accumulation its rate has a very close association with the etiological factors, as shown by Cabot. In a study of 50 cases the rate of fluid accumulation per day was: Cardiac weakness, 36 to 54 oz.; cirrhosis of the liver, 20 oz.; chronic nephritis, 13 oz.; solid tumors of ovary, 12 oz.; neoplasm of the abdominal organs and glands, 11 oz.; adherent pericardium (before cardiolysis) 11 oz.; adherent pericardium (after cardiolysis), 2 oz.; uterine fibroid, 8 to 11 oz.; tuberculous peritonitis, 5 to 6 oz.

The abdomen is filled with a fluid causing what may be called a painless distention and gives a dull sound on percussion, especially in the flanks. The dulness is movable, which is proven by the changing of the line of dulness as the patient occupies different positions. Inspection shows the abdomen distended with a smooth and shining surface, the base of the thorax is broadened, the navel more or less obliterated and the superficial veins enlarged. By palpation there is found a fluctuate, painless swelling of the abdomen with increased resistance in the flanks. The fluid can usually, but not always, be drawn off by aspiration. In cachexia and hydremia the fluid is slightly colored and of a milky appearance, clots slowly, specific gravity 1005 to 1015, and contains no cells. The specific gravity is usually lower than 1020 in chronic passive congestions and may contain 35 grams per litre of proteid. In carcinoma the fluid is turbid with cells, at times sanguineous and at other of a dirty greyish appearance, has a high specific gravity and coagulates spontaneously. The inflammatory fluid is straw or lemon yellow color, coagulates spontaneously, proteids 30 grams to the litre and specific gravity 1030 or above. It is possible in ascitic fluid to determine urea, uric acid, allantoin, zanthin, cholesterin and sugar. It has been the author's routine in recent years to x-ray the abdomen, employing a Bucky diaphragm, and he feels that if carefully studied the outlines of the peritoneal sac when it contains fluid, at least in many cases, may be observed. The central portion of the sac appears dense and the edges denser, and it is quite often semi-symmetrical, and while it is of no negative value it has confirmed cases in which fluid was determined by the ordinary physical signs. It would be fortunate indeed if the presence of fluid could be determined early. There must be many cases with even moderate amounts of fluid which escape the ordinary examination. In every case of edema of the feet and ankles the abdomen should be carefully examined.

Diagnosis.—The diagnosis must depend upon the history, painstaking physical examination, a cytologic examination of the fluid, and lastly an x-ray plate of the abdomen, (a Bucky diaphragm should always be used).

The diagnosis of fluid in the peritoneal cavity has been considered comparatively easy, the etiology less so, and yet reasonably plain. Rolleston has said, "The diagnosis of cirrhosis is a most difficult one." It is the early diagnosis we strive for and the most difficult to obtain, and like most diagnoses the late ones are apparent and comparatively easy to make. In the larger majority of cases the fluid in the peritoneal sac is serous and may be called the true ascites. The causes are usually mechanical, or a mildly inflammatory peritoneum. A dense exudate is found especially in cases of carcinoma or tuberculosis, while a sanguineous exudate is by no means rare. A purulent fluid usually follows superadded peritonitis, while a chylous arises from a rupture of the thoracic duct, or some obstructive pathology to the passage of chyle through the lymph into the receptaculum chyli. Anders states that of 128 cases of milky ascites many were chylous. The latter is due to the presence of epithelial cells, microscopic fat and degenerative pigment. Reviewing the etiology and the various pathological conditions under which fluid may accumulate in the peritoneum, we are struck in many instances by the lack of constancy in the varieties of fluid with a corresponding variety of pathology.

Differential Diagnosis.—*Ovarian Cyst.*—The enlargement is at first unilateral, but finally it becomes large enough to occupy the central part. The dullness may be well over the central abdomen, but the flanks are tympanitic. An x-ray examination was quite recently of assistance in distinguishing between an ovarian cyst and ascites.

Distention of the Bladder.—The history here is of value, and in all doubtful cases catherization should be performed.

Obesity of the Abdomen.—This is a condition in which one would hardly expect a mistake, and yet a succusion wave can be felt under some circumstances; however, there is no dullness in the flanks.

Tympanites.—There is no succusion wave or no changeable dullness. In dilatation of the stomach and intestines the barium meal and x-ray plate are of positive value.

Cysts of Abdominal Origin.—A cystic kidney rarely occupies both sides of the abdomen, and if on one side can surely be outlined by physical signs and x-ray plate, (Bucky diaphragm). A cystic liver or pancreas will require the same procedure. The history with physical signs and x-ray plate will probably diagnose an encysted exudate in peritonitis.

Pregnancy.—A large collection of amniotic fluid might be mistaken for ascites. It should give the history of pregnancy, and a careful vaginal examination should be made. In any case of suspected pregnancy it is unwise to use the x-ray exposure.

Choleperitoneum.—Barker describes biliary peritonitis, calling it choleperitoneum. Bile appears in the peritoneal cavity after a rupture of the bile ducts. This rupture may cause acute peritonitis if infected, but if not, inflammation need not result. The condition may follow a rupture of a hydatid cyst connecting the bile duct. Johnson and Wolfe report choleperitoneum associated with cholecystitis with perforation.

Hemoperitoneum.—In hemoperitoneum there is an enlargement of the abdomen without fever, and the diagnosis is based on the character of the fluid. It is described by both Barker and Cabot. Hemoperitoneum is pure blood in the peritoneal sac, and is distinguished from a sanguineous effusion. It is a sequela of extra-uterine pregnancy, trauma of the abdominal organs, ruptured aneurism, hemorrhage from a sarcoma or carcinoma, and rupture of varicose veins. Recently the author saw a case of hemoperitoneum result from an inflammatory adhesion which was punctured by a trocar. Faintness, pallor, dyspnea, restlessness and tachycardia, vomiting and colicky pains may be present in the abdomen, and even a collapse. The symptoms usually call for an immediate laparotomy.

Chylous Ascites.—Anders and Boston describe separately chylous ascites. It is classed under two types, true and false. The exciting factors are carcinoma of the peritoneum, tuberculosis, cardiac-vascular conditions, rupture of the thoracic duct, distention of the liver, puerperal sepsis, rupture of the receptaculum chyli, cysts and infection, and the filaria Bancrofti. It may occur under one year of age, but usually between thirty and fifty. Chylous ascitic fluid has a specific gravity of 1013, contains 3.06 per cent. of albumin, 2.08 per cent. of fat, and .32 per cent. of sugar. Microscopic chylous fluid consists of emulsified fats, pigment, fat globules and granular debris. The pseudo-chylous effusion contains a large number of epithelial cells in various stages of degeneration, a few fat droplets and granular debris. It collects slowly, sugar is absent, there is a great variation in size of fat droplets, and the fat is affected by diet.

Treatment.—**Rest** is a great factor, especially in cardiac and renal cases. **Diet** may play an important part, and at any rate it is one of the factors that can add to comfort. Each patient should have his food capacity determined, and his food tolerance tried out clinically. In the consideration of diet very naturally the causative factors receive primary attention, and the impaired physiology of the gastro-intestinal tract is to be considered. In addition to a food deficiency and lack of food balance these factors always must be considered. If we were to give a specific diet the author would suggest the **Karell method** with a **Mitchell modification.** In extreme ascites very little water should be taken. If the fluid diminishes the food may be increased, but it is a slow and tedious process. A glass of milk is given four times a day with zweibach. If milk disagrees substitute buttermilk, which often does better than sweet milk. It is a good plan to commence with 200 c.c. daily and increase this up to 3000 c.c. After the patient improves a soft egg may be added, and finally from three to six given daily. The diet is then shifted to a typical soft hospital diet; the food, however, should be salt poor. During all this time (some four to six weeks), if the ascites does not decrease, the fluid should be kept down to 800 c.c. daily. Friedenwald reports that cures and improvement can be brought about, and this is the author's experience. The improvement was in the heart and lowered blood-pressure, and the increase in urine passed. The chlorid output is greater than the intake. Loss of weight may be looked for, but that is not important for the time being. Of course if symptoms of uremia arise then the diet must be discontinued. Alcohol is forbidden. Lavage of the stomach and enemas usually do more harm than good. Undoubtedly some forms of ascites can be improved by cathartics and diuretics. The author employs **magnesium sulphate** or **jalap powder.** Beyond doubt the following prescription is of benefit, especially in cardiac and renal cases:

℞ Massæ hydrargyri.
Pulveris digitalis.
Pulveris scillæ aa grains xx (1.3 grams).
Dividi in pilvlas No. xx. M. Sig: One pill three times a day.

Another useful prescription is:

℞ Potassii citratis ℥ ss. (15.50 grams).
Infuse digitalis f ℥ vi. (178 c. c.).
M. Sig: One tablespoonful three times a day.

In 1909 Eichhorst spoke in the highest terms of the value of cream of tartar, stating that it prevents the accumulation and hastens the absorption of fluid:

Decocti althaeæ 180.0 c.c. (6.07 f. ℥).
Potassii bitartartis 15.0 grams (231.48 grains).
Syrupii simplicis 20.0 c.c. (5.42 f. ʒ).
M. Sig: Shake well. One teaspoonful every two hours.

We should not be pessimistic in our attitude toward our patients. Scrupulous attention to detail may give favorable results, personal factors and general management give much comfort, and the patients demand or look for someone who will give it. A well-established cirrhosis has not an anatomic cure, but it has a functional field for work. The fluid must be removed before the patient suffers from its presence. An early **paracentesis** is less objectionable than active purgation. There are a few dangers in paracentesis. The median line, a few inches above the symphysis pubis, is a suitable place. The bladder should be emptied, a small trocar should be used and strict antiseptic precaution taken. The skin should be sprayed with ethyl chlorid, a small incision made through it with a sharp knife, and the trocar is then plunged through into the peritoneal cavity. The fluid should escape slowly. After the removal of the trocar the wound should be painted with collodion, although a suture is preferable. After a removal of fluid a nine-tail bandage should be used, the pressure of this preventing hemorrhage from stomach and intestines. A careful physical examination should be made after the removal of the fluid. A rapid collapse following paracentesis should lead to the search for a hemorrhage. If digitalis is used before operation there is a little danger of a cardiac collapse. Two hundred and fifty cubic centimeters of normal salt may be given one hour before and twelve hours afterwards. Purgatives do more harm than good, but one of the agencies to stimulate peristalsis should be used, preferably the **cascara cathartic pill.** The **Talma operation** in a few selected cases may be of benefit, and should not be overlooked. It is needless to say that removal of fluid in hemoperitoneum and choleperitoneum should demand **surgical intervention immediately.** Tuberculous peritonitis as well as mechanical conditions which may cause ascites should be considered surgically. In tumors and growths of various kinds it is needless to say they should have surgical consideration. The hemorrhage from the stomach and intestines should have the usual treatment.

Prognosis.—Ascitic fluid may remain in the peritoneal sac for several years. Usually when it is pronounced it foretells fatality which occurs within a year. Rest, stimulation, tapping, and surgical intervention may modify ascites and prolong life. In a few cases surgical intervention may effect a cure. As might be expected in cardiac affections, anything which weakens the myocardium increases the fluid. Generally speaking, ascites is an unfavorable symptom, but does not positively presage a fatal termination. It should, therefore, have the benefit of treatment.

DISEASES OF THE OMENTUM

Omentitis.—The great omentum is frequently inflamed as a result of operations in the ileocecal region. Both the acute and chronic inflammation of the omentum are a part of or secondary to such general infections as tuberculosis and syphilis, and may be the seat of a carcinoma. As a matter of fact any infection may involve the omentum, but this does not often occur.

SYMPTOMATOLOGY.—The symptoms appear from a few days to several weeks after the operation. They are fever, pain, vomiting, constipation, and swelling. Occasionally there is a chill, then the temperature rises to 103° F. Vomiting may occur when cases commence acutely. Pain is always present, and is limited to the area of swelling. The location of the swelling is in the upper right or left quadrant or both, but it may extend to the midaxillary line or to the lower quadrants. The skin is doughy and pressure is painful. Probably all cases have some pus in the inflammatory mass. Cases, either acute or chronic, may appear insidiously, lasting for months. Adhesions may form which lead to intestinal obstruction.

TREATMENT.—Until the diagnosis of the inflammatory mass is definitely located hot packs or the ice bag are to be used. An operation is the real treatment when once the diagnosis is made.

Tuberculosis of the Omentum.—Tumor masses may form in the peritoneum. The diagnosis is made by the finding of tuberculosis in other organs. The omentum is first infiltrated and becomes retracted when it can be felt as a nodular mass in the epigastrium.

Carcinoma of the Omentum.—This usually occurs with an involvement of the colon. The most frequent site is the sigmoid flexure. Sooner or later symptoms of chronic obstruction appear.

DISEASES OF THE MESENTERY

Tumors in the mesentery are rare. The carcinous are usually found in separate masses and are located deeply beneath the intestines. Since they generally occur by metastases they can be recognized by the diagnosis of the primary growth. Tubercular tumors give the same physical signs, and they too are determined by the discovery of the disease in other organs. The glands of the mesentery are in most all cases the location of the primary inflammation. Any infection whatsoever, but particularly tuberculosis, may involve these glands. From them follows a chronic inflammatory mesenteritis, or perhaps more correctly stated, a mesenteric peritonitis. Inflammatory conditions of the mesentery impair peristalsis of its corresponding intestine, hence there is a marked constipation. At times the inflammation involves the sigmoid, attaching one end to the other, a condition which may cause volvulus. Chronic mesenteritis may be diffuse involving a large part of the small intestine. As a result of this it often leads to a local plastic inflammation of the peritoneum, thus causing constriction and adhesions of the bowels. A diseased mesenteric gland is the focus from which the inflammation extends to the mesentery, and then on to the peritoneum resulting in all the ill effects of an adhesive process.

The treatment is expectant, using such local measures as hot packs or the ice bag, and finally resorting to surgical interference if drainage is needed or mechanical repair indicated.

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IMPORTANT

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